A manual and atlas of medical ophthalmoscopy / by W.R. Gowers.

MEDICAL OPHTHALMOSCOPY

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OF

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BY

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PREFACE TO THE THIRD EDITION.

In preparing this, the third edition of "Medical Ophthalmoscopy," the whole work has been subjected to a revision sufficiently thorough to involve additions and alterations on almost every page and in almost every paragraph. An endeavour has been made to embody in it whatever of real value has been added to our knowledge, since the appearance of the last edition, and to present the facts to the reader in the aspect that they bear to the author, as viewed in the light of his personal experience. Accordingly, in many parts various statements have been not only added to, but recast in what will be found, it is hoped, a more practical form.

The microscopic figures that were represented on photo-lithographic plates in preceding editions have been re-engraved as phototype blocks, and appear, in this edition, in the text of the work, in connection with the subjects to which they refer. Other ophthalmoscopic figures, prepared in the same way, have also been added. The cases that were described in full in previous editions have served their purpose, and the extended and extending use of the ophthalmoscope in medicine has made such facts as they illustrated familiar alike to physicians and students. Brief epitomes have, therefore, been substituted, and placed in relation to the facts that the case illustrates. Instead of these, an account is given of the most convenient procedure in drawing the appearances that are seen in the eye. It is hoped

that these hints may, at least, have the effect of leading students to adopt a practice that will be found to be of great value, even beyond the subject to which it is applied.

I have had, in this edition, the help of Mr. Marcus Gunn, who has conferred on the work the advantage of a final revision, and has also superintended its passage through the press. To his knowledge and care the reader is largely indebted.

I may add the following extract from the preface to the first edition, published in 1879:—

"With one or two exceptions all the cases described and figured were met with in the course of purely medical work, chiefly at University College Hospital, and at the National Hospital for the Paralysed and Epileptic. In the preparation of the illustrations, great care has been taken to secure the utmost possible exactness. The autotype plates are reproductions of sepia drawings; and this method has been chiefly employed because by it a more exact representation of delicate pathological appearances can be obtained than by chromo-lithography. This method has also the advantage of fixing the attention on the changes of form, rather than upon the alterations in colour, which, important as they are, very often mislead the inexperienced. Chromo-lithography has been employed for some subjects in which the changes of tint are of predominant importance. It is intended that the autotype plates should be studied by the aid of the descriptions prefixed to them, and it is believed that, thus examined, those who are accustomed to the use of the ophthalmoscope will not miss the absent colours. With one or two exceptions," specified on p. 305, "the drawings were all made by the direct method of examination."

W. R. GOWERS.

50, Queen Anne Street,
July, 1890.

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INTRODUCTION.

The ophthalmoscope is of use to the physician because it gives information, often not otherwise obtainable, regarding the existence or nature of disease elsewhere than in the eye. This information depends upon the circumstance that we have under observation—1. The termination of an artery and the commencement of a vein, with the blood circulating in each. 2. The termination of a nerve, which, from its close proximity to the brain, and from other circumstances, undergoes significant changes in various diseases of the brain, and in affections of other parts of the nervous system. 3. A nervous structure—the retina, and a vascular structure—the choroid—which also suffer in a peculiar way in many general diseases.

For the efficient use of the ophthalmoscope in medical practice, the student must be familiar with the use of the instrument; he must also be familiar with the normal fundus oculi, with the changes in its appearance (congenital and other) that are of no significance, and also with those that are ocular in origin, such as posterior staphyloma, glaucomatous excavation, and the like. An acquaintance with these must be gained from the ophthalmic surgeon before inferences can safely be drawn regarding the significance of other alterations met with in various diseases. The following pages assume the possession of a general knowledge of the use of the instrument, but a few words

regarding some points which are of special importance may be of service.

A first requisite in medical ophthalmoscopy is familiarity with the direct method of examination. The disc is then seen magnified many times; and this method may show minute changes of the highest significance, which cannot otherwise be detected, or the true nature of appearances which, seen by the indirect method, are obscure. But both methods should always be employed. Not only has each its special advantage, but the two together often give information which neither alone affords.

Another requisite is skill in the examination without dilatation of the pupil. In most eyes much can be seen with the pupil undilated—often all that is necessary, and almost always enough to determine whether or not there is more to be learned by dilatation. The coincident paralysis of accommodation is a source of annoyance, and is especially resented by patients when there is no disease of the eye itself. If the sight has not been previously affected, it often happens that in brain diseases there is a subsequent failure of sight, due to changes, neuritis, atrophy, &c., which afterwards progressed. The failure of sight in such cases is often ascribed by the patient, not unnaturally, to the effect of the mydriatic.[1] For the same reasons one pupil only should be dilated at a time, unless the sight of both eyes is already impaired. If it is a matter of indifference which is chosen, an eye, the sight of which is impaired, should be chosen in preference to the other. These disadvantages have been lessened by the use of other mydriatics than atropine, or by using eserine when the examination is over, to contract the dilated pupil. Homatropine, however, has largely superseded atropine as a dilator, since the paralysis of accommodation passes off in a few hours, and the

1 "If we use the ophthalmoscope, or if we use atropine, or if we apply a blister to the head, or adopt any new kind of treatment, the patient may blame us for his blindness, if he saw well before such procedures. A patient who reads the smallest print and supposes his sight to be good, may have double optic neuritis. The use of atropine affects his sight for near objects

dilatation of the pupil seldom persists more than a day. Cocaine is likewise a useful mydriatic, on account of the short duration of its effects, and from the facility with which they yield to eserine. Its use is particularly indicated where there is any danger of exciting increased tension in the eye-ball by ordinary mydriatics.

In making an examination with the ophthalmoscope, it is best to look at the eye first from a distance, in order to ascertain whether the red reflection from the fundus is clear. This at once gives information regarding the presence or absence of opacity of the lens or vitreous, or may reveal iritic adhesions—conditions which convey important information, and explain what would otherwise be a puzzling obscurity of detail. Next, the refraction of the eye should be roughly estimated by observing if the vessels of the retina can be distinctly seen from a distance, and, if so, whether they move in the same direction as the observer's head (hypermetropia), or in the opposite direction (myopia). The knowledge of the condition of the eye thus gained is of much importance, since in myopia the details of the fundus appear, by the indirect method, small, and in hypermetropia they appear large. If necessary, the refraction may be more accurately ascertained by the use of a refraction ophthalmoscope: the lens needed to correct it, if the observer's refraction is normal, is the indication of the degree of error.

It is frequently necessary to examine patients in bed. The indirect method of examination can be applied as readily to a patient in bed as to one sitting on a chair, the

gravely, and if, from the advance of the neuritic process, what I may call retinal sight fails before the effect of the atropine has passed off, he very naturally blames us for the subsequent permanent affection of his sight. A patient, when asked how long his sight had been bad, replied, 'Only since the drops had been put in.' We must, then, when we discover neuritis, sight being good, tell the patient that his eyes are not really good, and that we are anxious about his sight. Whether we give this warning or not, we shall be blamed by an unintelligent patient for 'tampering with his eyes.' We must, however, act for our patient's good, regardless of selfish considerations. In very many cases we can see enough for diagnostic purposes without using atropine."—Hughlings-Jackson, Lectures on Optic Neuritis, "Med. Times and Gaz.," September 16, 1871.

most convenient place for the light being on the pillow above the patient's head. Even in daylight little difficulty is experienced unless the pupil is small, but the examination is facilitated by a screen of some kind, even by the shade of an umbrella. The direct method presents more difficulty; a convenient position is at right angles to the patient, with the lamp on the opposite side of the patient's head.

All who have employed the ophthalmoscope in medical practice will agree with Hughlings-Jackson in urging the routine use of the instrument in all diseases in which ophthalmoscopic changes are, even occasionally, met with. It often happens that unexpected information is gained regarding the nature of the disease, or its probable consequences.

It has been remarked that the medical ophthalmoscopist should possess familiarity with those changes in the eye which are of purely ocular significance. It is of equal importance that he should be familiar with those congenital changes in the eye which are of no significance. Many of these will be alluded to in describing the morbid appearances with which they are most liable to be confounded. One or two, which give rise to special trouble to the beginner, may be here briefly mentioned. One of these is the variation in the colour of the optic disc. It has been well remarked that the tint of the optic disc may vary as much as the tint of the cheek. It is always redder in the young than in the old. In the latter the redness has often a grey tint mingled with it. In the young the tint may even be scarcely or not at all paler than that of the adjacent choroid. When the choroid is bright in tint, the apparent redness of the disc is increased by indirect examination with a wide pupil and a bright light, and is a very frequent source not only of error in diagnosis but of scientific mistakes. It is the sharpness of the edge of the disc to which attention should be especially directed.

When the physiological cup is very large, the vascular portion of the disc is confined to a narrow rim at the side, often much narrower than that shown in Pl. III. 1, which represents a large but not very large cup. When the part of

the disc occupied by the nerve fibres is reduced to, say, one-half of that shown in the figure, the fibres are so crowded together that the choroidal limit is often less distinct than normal, and the central white cup may be mistaken for the disc, the edge being regarded as part of the fundus. Knowledge of this danger, however, will be sufficient to prevent an attentive observer from falling into this error; there is no confusion on direct examination.

White patches near the disc, due to choroidal atrophy and to opaque nerve fibres, sometimes present puzzling appearances (Fig. 1). The recognition of choroidal atrophy by the greyish-white tint of the sclerotic, by the pigment disturbance, and by the comparative absence of change in the retinal vessels, is usually one of the first points learned. Now and then a narrow posterior staphyloma may surround, or almost surround, the disc, and its edge may be mistaken for the edge of the disc, which then appears white with a red centre, an appearance with which I have known beginners to be much puzzled. (It is well to remember that posterior staphyloma may be seen occasionally

FIG. 1.—OPAQUE NERVE FIBRES,
Surrounding optic disc, and concealing the vessels in that neighbourhood.

in hypermetropic eyes, as well as in the myopic eyes, in which it is so common.)

The white patches of opaque nerve fibres (such as are shown in Fig. 1) are characterized by their position, adjacent to the disc; by the peculiar shape of the spot, which, if large, follows the course of the nerve fibres; by the partial concealment of the vessels; the feathery edge; and by the centre of the disc being commonly unconcealed. When a small patch lies near, but separated from the disc, the resemblance to an inflammatory exudation may be very close; the characters of its edge, and the absence of other changes, will usually enable its nature to be recognized.[1]

Peculiar white films sometimes lie in front of the vessels on the disc, looking like fragments of tissue paper or white gauze, and allowing the vessels behind to be dimly seen. These may be left by a pathological process, but they seem to be occasionally congenital, and caused by an undue development of tissue at the back of the vitreous. When congenital, the vessels are merely concealed; when pathological, they are constricted. In one case which came under my observation, a congenital film extended over the upper half of the disc, and ended on one side in a reflected edge.

In considering what may be learned regarding the conditions of the general system by observation of the fundus oculi, it will be convenient to consider, in the first place, specially those intra-ocular changes which are of general medical significance, viz., the changes in the vessels and the circulation; the changes, inflammatory and atrophic, in the optic nerve; and, more briefly, the alterations in the retina and choroid; and secondly, the changes which are met with in special diseases of the nervous and general systems.

[1] It is of great importance that the aspect of these opaque nerve fibres should be familiar. They sometimes give rise to curious errors in diagnosis. I was once taken to see a patient in whom a large and characteristic patch of this description was supposed to be of syphilitic origin, and to indicate that a cerebral affection, from which the patient was suffering, was of the same nature.

PART I.

CHANGES IN THE RETINAL VESSELS AND OPTIC NERVE OF GENERAL MEDICAL SIGNIFICANCE.

THE RETINAL VESSELS.

In no other structure of the body are the termination of an artery and the commencement of a vein presented to view, and information regarding the general state of the vascular system is often to be gained from an inspection of their size, texture, and the conditions of the circulation within them. It must be remembered, however, that the vessels there seen are of very small size. One of the primary divisions of the retinal artery, large as it appears to direct ophthalmoscopic examination, is in reality so small as to be scarcely visible to the unassisted eye, being less than the $\frac{1}{100}$th of an inch in diameter, and the smallest vessels visible with the ophthalmoscope are not more than the $\frac{1}{700}$th of an inch in diameter. But these, it must also be remembered, are considerably larger than capillaries. The retinal capillaries are always invisible, and, away from the optic disc, they are never so numerous as to occasion any recognizable reddish tint. The red colour of the fundus oculi is due to the choroidal vessels.

A second point to be remembered is that the red lines spoken of as the retinal arteries or veins are not the vessels themselves, but the columns of blood within them. The walls of the vessels are, as a rule, invisible; they are always invisible to the indirect method of examination, but by the direct method the walls of the larger branches may be sometimes seen, as fine white translucent lines along the sides of the red column of blood, most distinct where one vessel passes over another. They are best seen by feeble illumina-

tion, and especially by so moving the mirror as to render the illumination slight and oblique. Sometimes, as will be described immediately, the outer coat of the vessel is so thick as to be very conspicuous.

The paler line which runs down the centre of each vessel is probably a reflection of the light from the middle of the anterior surface of the column of blood. It is distinct only when the vessel lies in a plane at right angles to the line of observation. If the vessel, in consequence of an antero-posterior curve, ceases to be in a plane at right angles to the line of observation, this central reflection is no longer visible, and the whole width of the vessel is of the same dark colour as the edge. In the case of veins this change is very striking, and the greater amount of colour makes these portions appear darker in tint than the rest.[1] Many examples of this will be found in the appended plates, as in I. 4, II. 1, III. 2, 4, V. 5, 6, &c.

Size.—In estimating variations in size of the retinal vessels allowance must be made for the refraction, *i.e.*, magnifying power of the eyeball, remembering that, by the indirect method of examination, in myopic eyes the details appear small, while in hypermetropic eyes the objects appear large. In the direct method there is less variation, because, for distinct vision, the myopic refraction requires correction by a lens. The apparent size of the disc may be taken as the guide to the amount of magnification. There is no very exact method of estimating the absolute size of the vessels.[2] Sometimes, however, the alteration is such as to be at once evident and unquestionable. A little custom will enable a distinct deviation from the normal to be readily recognized.

[1] It is probable that such portions of the veins are especially dark, since, by their obliquity to the line of vision, this passes through a greater amount of blood ; the light reflection from behind is thus lessened, the choroid being much paler than the blood in the veins. Hence the change in tint is far greater in the veins than in the arteries, which are nearly of the colour of the choroid.

[2] If a wire grating is fixed in front of the light used for the direct examination, the lines of the wires are seen on the fundus, and can be used for measurement. An instrument for use with any light, with wires a definite distance apart, is described in previous editions of this book.

Special attention must be given to the number of primary branches of the vessel. It often happens that veins are thought to be pathologically large, merely because they are few.

The relative size of the arteries and veins can be observed with more exactness than their absolute size. In comparing the two it is usually desirable to have the pupil dilated, since the vessels have often to be traced for a considerable distance from the disc. A difficulty arises from the fact that the distribution of the arteries and veins corresponds approximately, but not exactly. Sometimes two arterial branches accompany one venous trunk: sometimes two veins accompany one artery. But in each eye there is usually at least one set of vessels which have a nearly identical course and distribution, run side by side, and are available for comparison. When this is the case it will be found that, as a rule, the width of the artery is about two-thirds or three-quarters that of the vein. An alteration in this relation may arise from a change in the size of the artery or of the vein. The change may be so considerable that its nature is at once evident: *e.g.*, the veins may be obviously wider than normal, or the artery unquestionably narrower, perhaps visible as a mere line even by the direct method of examination (Pl. IX. 4, XII. 2, 3). When the difference is slighter, we have to form an opinion as to the change on which it depends (whether enlargement of vein or diminution of artery) by our knowledge of the normal size of the vessels—an approximate absolute estimation. A little familiarity with the appearance of the vessels under normal conditions will commonly enable an opinion to be formed as to the direction in which the change exists.

Equality in size of the artery and vein is usually due to dilatation of the artery. When the relative size of the artery is smaller than that given ($\frac{3}{4}$ or $\frac{2}{3}$) it is generally due to one of three causes: (1) Venous distension, general or local; (2) Imperfect filling of atonic veins, in consequence of which they are flattened at right angles to the line of observation: (3) Contraction of the arteries, which may occur from general anæmia (in which case the veins are large

and atonic) or from primary arterial contraction, as sometimes in Bright's disease (in which the veins also are commonly small), or from local obstruction to the entrance of blood.

Veins.—Increased width of the veins, therefore, usually means their dilatation, either from distension or from atony, and this effect is commonly uniform. The central reflection is preserved in normal characters. A varicose condition has been observed in a few cases, but is of doubtful significance. A remarkable example of moniliform dilatation has been figured by Liebreich in his Atlas. The distension may be part of a general venous fulness, as in cases of cardiac or pulmonary obstruction; or it may be of local origin. Increased intra-cranial pressure of *rapid* development, probably causes at least a transient increased fulness of the retinal veins. When of *slow* development, this effect is rare, in consequence of the anastomoses of the orbital and facial veins. Thus the veins may become large in acute, and not in chronic, hydrocephalus. The same effect may be, it is commonly believed, the consequence of distension of the sheath of the nerve, and of pressure within the sclerotic ring. The former will be considered in connection with neuritis; its precise influence is difficult to estimate. The influence of the rigid sclerotic ring cannot be regarded as demonstrated beyond question. It is said to intensify the effect of an obstruction, but the evidence is hypothetical. A very efficient cause of distension of the veins is their compression by inflammatory products within the optic papilla. Extreme distension occurs also in cases of thrombosis in the central vein of the retina behind the globe.

Increased width of vein, however, does not necessarily imply over-distension. A vein which is underfilled may present an increased width. Usually, if the quantity of blood within a vein is less than normal, its contractile power enables it to adapt itself to the diminished bulk of the contents; it retains its cylindrical form, and both appears and is narrower. But in states of anæmia, the atony of the vein may prevent it from following the contents in calibre,

and retaining the cylindrical form. It may then have a more or less elliptical lumen (the same circumference enclosing a smaller area as an ellipse than as a circle), and in the retina, in consequence of the intra-ocular pressure, the flattening always takes place in the plane of the retina, at right angles to the line of vision, and the vein appears of undue width. At the same time the central reflection is altered, becoming commonly indistinct, but sometimes unduly broad.

This condition of the veins is seen especially in extreme anæmia, and in leucocythæmia, as in the accompanying figure (see also Pl. XI. 1 and 2). In these cases the arteries are usually smaller than normal, and so the contrast between the veins and arteries is enhanced.

Diminution in the size of the veins is probably always the result of diminished supply of blood.

The arteries may be diminished in size by causes similar to those which lead to increased width of the veins, such as local obstruction to the entrance of blood. The latter does

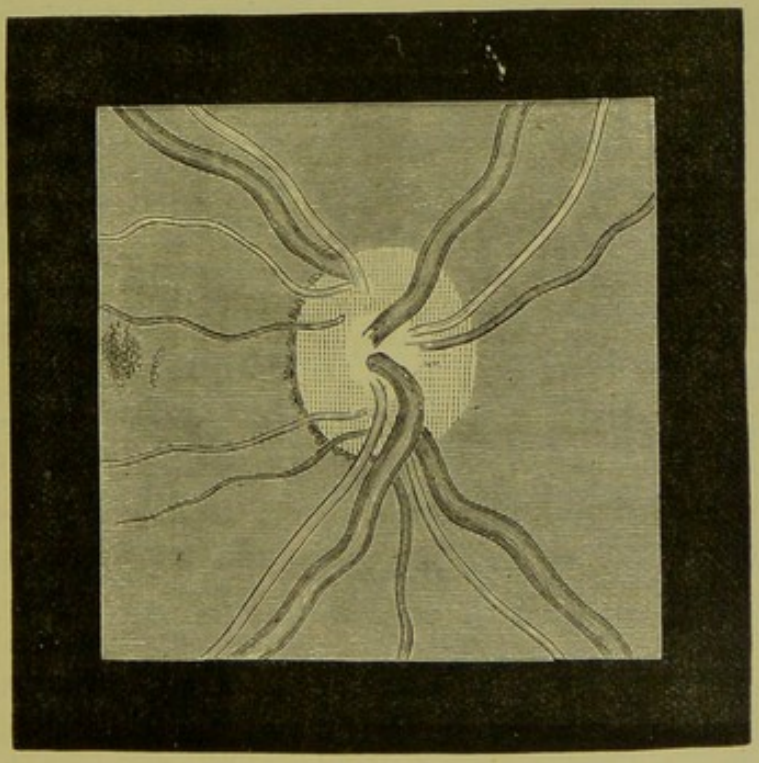


FIG. 2.—BROAD RETINAL VEINS AND NARROW ARTERIES.
From a case of leucocythæmia.

not appear to result from general intra-cranial pressure, probably because of the resistance afforded by the strong walls of the arteries. It is doubtful whether effusion into the sheath of the nerve is capable of diminishing the blood supply. It is certain, however, that the pressure of inflammatory products within the papilla, and especially their cicatricial contraction, may cause sufficient constriction of the artery to lead to a great diminution in the size of its branches. Hæmorrhage around the vessel, or the pressure of growths, may have the same effect. In no condition, however, does the diminution in the size of the vessel reach such a degree as in obstruction by embolism (Pl. XII. 2 and 3). General underfilling of the arterial system, as in cholera, may lead to a great diminution in the size of the arteries, their strong muscular coat maintaining their adaptation to the blood within them. Mere atony does not cause the increase in width in the arteries which is observed in the veins, because persistent spasm of the arteries is capable of causing a permanent diminution in their size. I have repeatedly observed this narrowing, especially in cases of Bright's disease, in the branches beyond the disc. It reaches its height when papillary obstruction is superadded, and then a degree of diminution in the size of the arteries may be seen, rarely if ever observed in obstruction from neuritis without kidney disease (see Pl. IX. 3 and 4). Two remarkable cases observed by Ramorius suggest that spasm of the retinal arteries may be a consequence of malarial poisoning (see Part. II., "Malarial Fevers").

Dilatation of the arteries is due to a vasomotor influence, and is conspicuous in some cases of exophthalmic goitre, in which over-action of the heart is superadded. It is doubtful whether the latter cause alone ever produces visible dilatation of the retinal vessels.

Arrangement.—The anatomical arrangement of the vessels varies considerably in different individuals, and is, in itself, of little medical significance. The number of branches into which the primary trunk divides, and the

number of tributary veins, should be noted in connection with the apparent size of the vessels. There is one point, however, which does possess indirect medical significance. The general arrangement of the vessels in the two eyes is usually similar. Moreover, similarity in vascular arrangement may be inherited. I have seen, for instance, a peculiarity in the course of the retinal vessels in a mother exactly reproduced in the eye of her daughter. This is a striking proof of the transmission of vascular arrangement in general; upon this depends the vascular strain, and, in part at least, the occurrence and locality of vascular degeneration, and even of vascular rupture. Thus, inspection of the retinal vessels suggests to us one way in which a tendency to cerebral hæmorrhage, or softening from atheroma, may be inherited.

Course.—The course of the retinal vessels usually presents few tortuosities, and those which exist are lateral, in the plane of the retina. A considerable increase in tortuosity may be associated with a nævus of the adjacent part of the skin.[1] The arteries are rather more tortuous in hypermetropic eyes than in others. When the vessels are elongated by their distension or atony, these lateral curves are exaggerated. Antero-posterior curves, at right angles to the plane of the retina, are indicated by the change in the central reflection already mentioned, by the relative displacement of parts at different levels on movements of the observer's head, and sometimes by slight obscuration of the vessel at the lowest point of the curve. They always indicate irregularities in the retina in which the vessels lie, commonly swelling, as in retinitis and retinal œdema.

Structural Changes.—Most changes in the tissue of the retinal vessels are visible only to the direct method of examination. The commonest change is an increase in the

[1] See Allen Sturge in "Clin. Soc. Trans.," vol. xii. 1879, p. 162. For cases of idiopathic tortuosity of retinal vessels, chiefly affecting the veins, *see* Benson, "Trans. Ophth. Soc.," vol. ii. p. 55; Nettleship, *ibid.* p. 57; Stephen Mackenzie, *ibid.* vol. iii. p. 101; all with accompanying drawings.

amount of tissue of the wall, especially of the outer coat, so that the red column of blood is bounded by distinct white lines. Such an appearance may be seen in most cases in the healthy fundus near the centre of the disc. At the point at which the vessels emerge from the disc they are, the arteries especially, often surrounded by this white tissue, sometimes like a little cloud upon them, and from it prolongations may be traced along the chief vessels. When a vessel curves over the edge of a hollow central cup, and is seen foreshortened, the white tissue of the wall often appears as a ring around the blood-column. When a disc is very full coloured, whether normally or from pathological causes, this white tissue is rendered by contrast very conspicuous, and may easily be mistaken for a pathological condition (Pl. I. 2). The difficulty is increased by the circumstance that it is sometimes a morbid appearance, left by preceding inflammation. In this case, however, it is usually accompanied by distinct constriction of the vessels, and it often extends along them beyond the limits of the disc. It has been thought that this tissue is sometimes a result of chronic congestion of the disc, insufficient to cause such an "exudation" as shall distinctly constrict the vessels. This is possible, but the condition is so common without either congestion or inflammation, that the presence of this appearance alone deserves little weight.

An undue visibility of the wall of the vessel is said to be sometimes caused by a "sclerosis" of the middle coat, a condition of thickening of the coat which, under the microscope, bears considerable resemblance to the appearance presented by lardaceous degeneration.

In very rare cases, there is such a thickening of the outer coat of the vessel, or an increase in its perivascular sheath, that the tissue is visible, not merely at the sides of the vessel but in front of it, concealing the red reflection from the column of blood within it, and broad white bands then indicate the position and course of the vessel. These bands may cease suddenly, so that lengths of red blood may alternate with the white bands. This condition has been seen

in Bright's disease, and a well-marked example is shown in Pl. XII. Fig. 1; it is then perhaps similar to the fibroid thickening around the vessels found in other organs. Sometimes a vessel may be narrowed at the affected area; more commonly its calibre is unaffected. In the case figured it is seen to affect the arteries only.

In most inflammatory conditions, leucocytes accumulate in the perivascular sheaths, and in the retina they may give rise to an appearance similar to that just described; this has been termed "perivasculitis." According to Liebreich, by a careful comparison of the relative width of the column of blood and of the white band, an opinion may be formed of the position of the new tissue, whether in or outside the wall of the vessel.

Fatty degeneration of the vessels is sometimes met with as a senile change, or after inflammation. It affects chiefly the outer coat, but has only been recognized by microscopical examination, and there is doubt whether it can be detected during life.

In senile fatty degeneration of the outer coat of the retinal vessels, calcification of the degenerated portion has been found after death. Actual atheroma—*i.e.*, endarteritis deformans—has not, so far as I am aware, been found in the retinal vessels after death; and in cases in which it is well marked elsewhere I have often looked for appearances in the retina suggesting its existence, but without success. The retinal arteries are far below the size in which atheromatous changes are common. They have been said to present undue tortuosity in this condition.[1]

Aneurism.—The retinal arteries are occasionally the seat of aneurismal dilatation. Instances of it are, however, rare, probably on account of the support which is afforded to the vessels by the vitreous humour. When aneurism does occur, its significance is important, because in no other way can the existence of aneurisms on vessels so small as those of

[1] Concerning so-called "Arteritis obliterata," *see* Fürstner, "Centralbl. f. Nervenkr.," 1882, and "Centralbl. f. Augenheilk.," 1882, p. 509.

the retina be ascertained. Dilatations of such small vessels are commonly not associated with aneurisms on large arteries, but when minute aneurisms exist in the retina they almost always exist also in the small arteries of other organs.

Two forms of aneurisms have been observed: (1) aneurisms of some size on the primary branches of the central artery on the disc: (2) miliary aneurisms of the arterial twigs in the retina, and of the small capillary vessels.

(1). Very few instances of the larger aneurismal dilatations are on record. One, which was described by Sous,[1] occupied the upper two-thirds of the disc, was oval in form, and presented distinct pulsation, synchronous with the radial pulse. The arterial branches in the retina were very narrow. The patient was a woman, aged sixty-four.

(2). Miliary aneurisms were found post mortem by Liouville,[2] in cases in which cerebral hæmorrhage resulted from the rupture of similar aneurisms in the brain. The largest was about the size of a pin's head; they were chiefly situated at the branchings of the vessels. In one case they were widely distributed through the body, being found on the minute arteries of the pericardium, mesentery, &c. They are frequently found in glaucomatous eyes. I have seen them during life on small arteries in a case of Bright's disease, in which there was extensive cardiac and vascular disease (Pl. XII. 1). The lower branch of the artery is seen to present three globular dilatations in its course, the third being just in front of a narrowed segment. The general characters of these aneurisms are there seen. The central reflection of the artery is widened at the dilatation in accordance with the altered surface of the blood within the vessel. The wall of the aneurism is, of course, invisible, just as is the wall of the vessel elsewhere; its existence is declared by the change in the form of the column of blood. Bouchut[3] has figured two examples of a series of fusiform dilatations of the retinal arteries in general paralysis of the insane. His figures, however, suggest considerable exaggeration.

[1] "Ann. d'Ocul." 1865, liii. p. 241. [2] "Comptes Rend." 1870, lxx. p. 498.
[3] "Atlas d'Ophtalmoscopie Médicale et Cérébroscopie."

The recognition of these minute arterial aneurisms presents little difficulty. The contours of the arteries must be followed from the disc to the ora serrata by the direct method of examination. A twist in a vessel may cause the appearance of a local bulging which may look like an aneurism, but a careful examination will prevent error. Minute hæmorrhages in the course of the vessels can be readily distinguished from aneurisms by the irregularity of the outline of the clot. Aneurisms, as a rule, contain fluid blood, and present a bright central reflection, which is absent in the extravasation. It must be remembered, however, that a miliary aneurism has been found surrounded by a halo of extravasation. The centre of any hæmorrhage situated at the bifurcation of a vessel should therefore be carefully scrutinized. A very rare condition has been figured by Galezowski, which might easily be mistaken for multiple sacculated aneurisms; it consists of numerous minute secondary gliomatous growths, connected with the retinal arteries. Some, however, were of large size, and unconnected with the vessels, and none presented any visible reflection.[1]

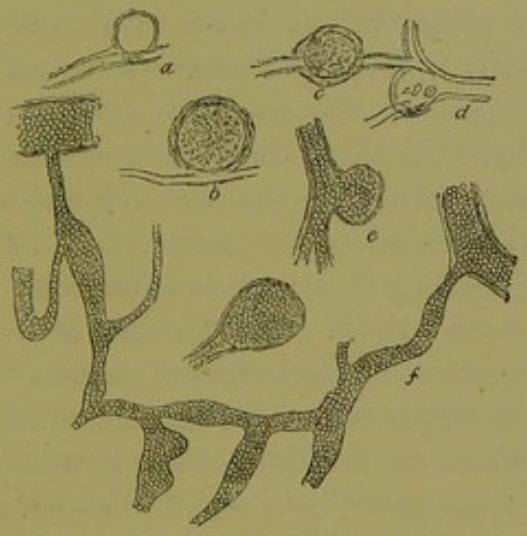


FIG. 3.—CAPILLARY ANEURISMS, AND VARICOSE CAPILLARIES.

a—*e* From a case of diabetes with retinal hæmorrhages (from preparations by Mr. Nettleship). At *a*, *b*, and *e* the aneurisms are situated laterally, at *c* in the course of a capillary, and at *d* at the bifurcation of a vessel (× 150). *f*, Varicose capillaries from a case of Bright's disease (× 150).

[1] In the "Trans. Ophth. Soc.," vols. iii. p. 108, and vi. p. 336, a striking instance of aneurismal dilatations of retinal arteries and veins is recorded by Story and Benson. The case affords a valuable illustration of the manner in which aneurisms may result from damage done to the walls of small vessels, by an inflammation of the walls as part of a general inflammation of the structures in which the vessels lie. The history of the case is unfortunately defective, but it is possible that the primary affection of the retina was syphilitic, and syphilis is known to be a cause of aneurisms of the cerebral arteries.

The retinal capillaries may present aneurismal dilatations sacculated in form, and also varicose dilatation. Examples of these are represented in Fig. 3, from a case of glycosuria described by Dr. Stephen Mackenzie.[1] Hæmorrhages into the retina and vitreous were observed during life. Capillary aneurisms, from a case of Bright's disease, are also shown in the same figure.

Changes in the Circulation.

The central artery of the retina brings blood to the eye from within the cranial cavity; the blood comes from an artery which also supplies part of the cerebrum and meninges: the retinal vein returns the blood chiefly to a cranial sinus. Hence the intra-ocular circulation has been regarded as a portion of the cerebral circulation, as participating in the same influences, and presenting the same modifications. This is, no doubt, true to some extent. At the same time it is probable that the consequences of the common origin of the cerebral and ocular blood-supply have been exaggerated. It is important to bear in mind that the intra-ocular circulation is peculiar in its rigid enclosure in a small chamber, in which it is always exposed to a certain amount of elastic pressure. Moreover, the anastomosis between the orbital and facial veins tends to prevent a close correspondence between the intra-cranial and intra-ocular veins. The relation between the cerebral and ocular circulation is unquestionably greatly modified by these and other influences.

Pulsation.—*Arterial.*—As a rule, before reaching arteries so small as those of the retina, the pulse-wave has become so feeble, the current so equable, that visible pulsation can no longer be perceived. The pulsation is also diminished by the normal pressure within the eye; this, in giving support to the retinal vessels, necessarily lessens their distension. If, however, the current be rendered less equable by an increase in the disproportion between the continuous flow and the intermitting wave, arterial pulsation may sometimes be

[1] "Ophth. Hosp. Rep.," December, 1877.

perceived. Locally, this disproportion may be increased by a change in the intra-ocular tension: thus a temporary distinct arterial pulsation usually results from the artificial production of increased tension by pressure on the globe with the finger. Again, a diminution of intra-ocular tension may, perhaps, sometimes occasion visible arterial pulsation.[1]

In conditions of acute anæmia from hæmorrhage, the continuous flow of blood into the small vessels may be feeble, and the pulse-wave then becomes distinctly visible.[2] But it is especially when the pulse-wave is increased in strength and suddenness that it becomes visible in the retinal arteries. This increase is developed in aortic regurgitation, and in that condition spontaneous pulsation of the retinal arteries is especially frequent, as Quincke,[3] Becker,[4] and Fitzgerald,[5] first pointed out. It is more distinct, the greater is the hypertrophy of the left ventricle, and is absent only when the heart is greatly weakened, when much aortic constriction coexists, or the amount of regurgitation is small. It may be seen best in the vessels on the disc, but can often be recognized far towards the periphery of the retina, and in this latter respect is distinguished from the pulsation due to mere increase of intra-ocular tension. It consists, like the pulsation of other vessels, in a widening and an elongation. The widening is best seen behind a division at a considerable angle, and is best recognized by attending to the central reflection. The elongation of the vessel is best seen where an artery lies in an S curve, especially towards the periphery, or when it forms a curve along the edge of the disc (Becker).

[1] Such a diminution is said sometimes to occur in the course of typhoid fever, and pulsation has been observed in the retinal artery in this disease by Schmall. See "Retinal Circulation and Arterial Pulse in General Disease," "V. Graefe's Archiv.," xxxiv. 1, p. 37, and "Oph. Rev.," 1888, p. 268.

[2] An arterial pulse has also frequently been observed by Schmall in cases of chlorosis. Here, as in anæmia generally, Rählmann ascribes the pulsation to hydræmia, but Schmall considers it due to "a certain amount of cardiac contraction, combined with sudden relaxation of the heart muscles, occurring in certain states of low arterial tension." (*Op. cit.*)

[3] "Berlin Klin. Wochenschr.," 1868, No. 34, and 1870, No. 21.

[4] "Arch. f. Ophth.," xviii. 206—296.

[5] "British Med. Journal," Dec. 23, 1871, p. 723. Dr. Stephen Mackenzie has also recorded several cases ("Med. Times and Gaz.," 1875, vol. i.).

In a case of supposed aneurism of the arch of the aorta, Becker found marked pulsation in the left eye, while in the right only a trace of pulsation could with difficulty be detected.

Pulsation in an extreme degree appears to be sometimes physiological. It was present in a man under my care who had also a very faint diastolic basic murmur but no hypertrophy or dilatation of the left ventricle, so that there was certainly not enough aortic regurgitation to account for the pulsation. A capillary pulse could readily be obtained in the forehead. The increased pulsation seemed to be in the small arteries only, since at the wrist, even when the arm was raised, the artery had the normal pulse-characters. In the eye, pulsation was conspicuous in both arteries and veins, and slight pressure on the globe increased it to such an extent that some veins on the disc, of full size in the diastole, actually disappeared at each systole. Moreover, the diastole of the arteries corresponded to the systole of the veins, and the pulsation in the latter must therefore have been due to the mechanism to be presently mentioned.

Capillary pulsation has been described in aortic regurgitation—a pulsatile redness of the disc—due to the intermitting distension of the capillaries in consequence of the great fall of pressure between the successive pulses. Such an appearance is, however, very rare, and can seldom be detected even when a pulsatile blush is visible on the forehead.

Venous.—Pulsation in the retinal veins may frequently be observed as a normal condition, chiefly in the large branches upon the optic disc,[1] especially where the veins curve down the sides of the cup. It is almost constant in aortic regurgitation, and is much more frequently conspicuous in this disease than the arterial pulse.

Several explanations have been given of the venous pulse. The theory which is, perhaps, the most plausible explains the pulsation by supposing that where the artery and vein are near together, in the sclerotic ring or optic

[1] Messrs. Lang & Barrett found a venous pulse on the disc in 73·8 per cent. of the eyes examined by them at Moorfields. "Ophth. Hosp. Rep.," vol. xii. p. 60.

nerve, the arterial distension compresses the vein and causes a temporary obstruction to the return of the blood. The nearer the two are, the more readily will this effect be produced, and the more slight a morbid increase needs to be for the artery to transmit an inverse pulsation to the vein.

Coccius suggested that the venous pulse depends directly on the intra-ocular tension, being analogous to that which occurs in glaucoma, and may be produced artificially by pressure on the eyeball. Every time the pulse-wave reaches the intra-ocular arteries, their distension causes a sudden increase in the intra-ocular tension, which compresses most the thinner walled veins, and lessens the amount of blood in them. Hence the contraction of the veins should correspond to the arterial diastole, to the pulse-wave, and the dilatation of the veins to the arterial systole, to the interval between the pulse-waves. As a rule, however, this is not the case: the distension of the veins nearly corresponds in time with the arterial distension. Hence, Stellwag von Carion imagined that the extension of the sclerotic by the increased intra-ocular pressure at each pulse stretches the lamina cribrosa, and narrows its meshes so as to compress the vein.

According to Donders, the increased intra-ocular pressure acts directly on the venous trunks in the optic disc, hindering the return of blood. Similarly, Jacobi, on the grounds of the common limitation of pulsation to the papilla, suggests that the increased intra-ocular pressure, depressing the papilla, augments the curve of the veins, and so causes a sudden obstruction to the circulation through them.

Helfreich, on the other hand, considers that the venous pulse is due to a pulse in the cerebral veins, grounding his opinion on experiments that show the tension in these to be high, and that they pulsate. He states that the venous pulsation is synchronous with the cardiac diastole, and that it is seen only on the disc, because of the firmer support of the veins away from the disc.[1] It has, however, been mentioned

[1] Ophth. Congress, Heidelberg, 1882, and "Ophth. Review," 1882, p. 408.

(p. 18) that the physical conditions in the eye are not exactly similar to those in the brain. Helfreich's theory seems to account for the coincidence of arterial swelling and venous swelling, but so also does the juxtaposition of the arteries and veins within the sclerotic ring mentioned above. If Helfreich's theory be correct, should not the pulsation be an invariable thing?

Putnam and Wadsworth (of Boston, U.S.A.) have described[1] an intermitting variation in size of the retinal veins, occurring independently of the pulsation, synchronous with the heart's action, and having a period of about five respirations, *i.e.*, about that of the variations in arterial tension found to occur in animals. Their observations have not yet been confirmed.

Anæmia of the Retinal Vessels may be part of general anæmia, or may be due to local pressure upon the artery, and transient anæmia is probably sometimes due to the vasomotor nerves.

When due to local causes ("retinal ischæmia" of the Germans) there is usually simultaneous pressure on the retinal vein, which runs side by side with the artery. The arteries are then unduly narrowed; whether the veins are distended or not depends on the rapidity or slowness with which the obstruction is developed. This condition is constantly seen during the contraction of inflammatory tissue in the papilla. In rare cases, in which the pressure is on the artery immediately after its entrance into the optic nerve, and behind the vein, which enters a little in front of the artery, the arteries may be narrowed without any distension of the veins.

Spasm of the retinal vessels has been supposed to occur in epilepsy, and also to be the cause of "retinal epilepsy," *i.e.*, epileptiform amaurosis. I have examined the retina in many cases of epilepsy immediately after fits without observing any marked change in the arteries. During several epileptiform convulsions, I have kept an artery and vein in view throughout the fit, by the direct

[1] "Journal of Nervous and Mental Disease," October, 1878.

method of examination, but have seen no change in the artery. The vein was distended during the cyanotic stage.

General defective blood-supply is much less evident in the vessels of the eye than elsewhere: probably because the intra-ocular tension effects a regulation of the size of the retinal vessels (Donders). Loss of blood, for instance, causes but a slight change in the retinal vessels, except an increased disproportion between the arteries and the veins, due in part to contraction of the arteries, and in part to atony and flattening of the underfilled veins. The effect of hæmorrhage on the size of the vessels soon passes off, because the volume of the blood is quickly reproduced by the passage into, and retention in it, of liquid from the tissues and alimentary canal.[1] A similar condition of the retinal vessels to that seen in acute anæmia was observed by v. Graefe in cholera. During the stage of collapse the arteries became narrow, the veins dark, but of normal width. Spontaneous pulsation appeared in the arteries, and was attributed to cardiac weakness, but may, perhaps, have been due to the diminution of the volume of the blood, rendering the amount ejected from the left ventricle at each systole so small that the shock (pulse-wave) predominated over the movement of the blood.

The acute cerebral anæmia of syncope is probably attended by a similar condition of the retina, and to it the transient blindness which sometimes succeeds syncope may be due.

Conditions of general defective blood-supply render the disc paler, but the variations in the tint of the disc, under physiological conditions, are so great that it is only by comparison of the state of the disc with its appearance in the same patient at another time, that any information can be gained from it. The other eye is usually affected in the same degree, and is not, therefore, available for comparison.

[1] In some observations on the effect of venesection in the corpuscular richness of the blood, kindly made for me by Mr. W. S. Tuke, on some patients of Mr. Wharton Jones, it was found that the fall in the number of blood-corpuscles indicating the dilution of the circulating blood to reproduce its volume, took place in the course of an hour. It was found also that the fall was greater than the amount of blood lost could account for—*i.e.*, that the hydræmia became for a time excessive, a fact which may account for the reputed influence of slight, quick loss of blood.

Hyperæmia of the Retinal Vessels.—(A.) *Active Congestion.*—Apart from the active congestion of commencing inflammation and of purely ocular conditions, such as refractive asthenopia, and exposure to excessive light, &c. (which are not considered here), an increased supply of blood to the retina may be due to whatever causes an overfilling of the whole or part of the arterial system of which the retinal artery forms part. Of these, excited action of the heart is the most potent. The retinal arteries may be seen to be large, and sometimes, though rarely, to pulsate, and the communicated pulsation in the retinal veins may also, commonly, be observed. When the overaction is long-continued, hæmorrhages may occur. A similar overfilling may occur from obstruction in another region of the internal carotid. Dilatation of the arteries, as in exophthalmic goitre, may also cause active hyperæmia.

(B.) *Passive Congestion.*—Passive congestion of the retinal vessels may occur from local or general causes. The causes of local obstruction to the return of blood from the eye are, for the most part, the same as those of local arterial anæmia. The most intense passive congestion ever seen is met with in thrombosis of the retinal vein. Pressure on the cavernous sinus only causes transient passive congestion of the retinal veins, on account of the free connection of the orbital and facial twigs. Passive congestion from general causes is very common, and results from whatever hinders the return of the blood from the head, or obstructs the circulation through the chest. The congestion of the eye is thus part of a cephalic congestion, or of a general venous stasis. The former commonly results from pressure on the jugular or innominate veins. The general congestion is the result of some pulmonary or cardiac obstruction, acute or chronic. The common acute causes are—cough, effort, and an epileptic fit. The ophthalmoscope shows the retinal veins in these conditions to be greatly distended. Unless, however, there is also disease of vessels, hæmorrhages rarely occur, no doubt in consequence of the support afforded to the vessel by the vitreous humour. The intra-ocular tension, and therefore

the external support, is probably augmented during these conditions of increased strain, in consequence of the fulness of the capillary vessels. It is true that the most intense congestion, such as that of suffocation, sufficient to cause death, does usually lead to retinal hæmorrhages, but minor degrees of congestion rarely do so unless the vessels are diseased. It is very common, for instance, for a violent cough, or an intense asphyxial stage of an epileptic fit, to cause rupture of a subconjunctival vessel, and a consequent extravasation, but it is extremely rare for any retinal vessel to give way. I have often, in such cases of epilepsy, searched the retina for extravasation, but the search has always been unsuccessful. In whooping-cough, retinal extravasations have been seen only in extremely rare cases.

Chronic general causes of passive congestion are chiefly heart disease (especially mitral) and emphysema of the lungs. In the general venous distension of congenital heart disease—cyanosis—the retinal vessels participate, often conspicuously. The venous distension may be extreme, and may be accompanied by normal arteries, or the arteries may be also large. The blood in the arteries and veins may be abnormally dark. Sometimes the retinal tissues are thickened. The congestion from emphysema of the lungs, and from dilatation of the right heart, is also often very marked. The retinal veins become much distended and tortuous, and the smaller branches, ordinarily invisible, may become conspicuous.

Hæmorrhage.—Rupture of retinal vessels and consequent extravasations of blood are very common in many morbid states, and are frequently of important general significance. They may occur as part of inflammation of the retina, and such cases will be considered subsequently. More frequently they are dependent directly on general conditions, or on retinal disease consequent on general conditions.

They vary much in size, number, position, and aspect. They may be so small as to be visible only as a spot or line on direct examination, or they may be three or four times

the diameter of the optic disc. There may be only one or two, or innumerable extravasations may exist over the whole fundus. When few they are commonly seated near the disc or in the neighbourhood of the macula lutea; when numerous, the largest are often situated near the macula. They often follow the course of vessels, especially the veins, but not unfrequently the arteries. Their shape and aspect depend very much on their position in the substance of the retina. The commonest seat is in the layer of nerve fibres. The fibres are separated, not torn, by the extravasation, and the blood lies between them, extending along their course in the direction of least resistance. Hence the smaller hæmorrhages are linear, the larger striated in part or altogether, and they often radiate from the disc. Such hæmorrhages are shown in Pl. V. 4, VI. 1, IX. 1, 2, X. 1, XI. 1. The next most frequent seat is in the inner nuclear layer. Here there is no tendency to striation; the extravasations are round or irregular (as in Pl. VI. 4, XII. 1). If the extravasation in this position is large it may separate the retina from the choroid, while a hæmorrhage in the nerve-fibre layer may break through into the vitreous. This sometimes happens in Bright's disease, as in one case which came under my observation.[1] The patient, a girl of seventeen, was admitted under Sir Wm. Jenner, suffering from chronic Bright's disease and hemiplegia. On admission there was well-marked albuminuric retinitis of the usual type. A fortnight later, a hæmorrhage occurred, partly obscuring the fundus. It did not become diffused, but remained attached to the retina by a pedicle.

Now and then, especially in the neighbourhood of the macula lutea, the blood may be extravasated in a thin film between the retina and the vitreous. Such an extravasation is commonly very irregular in shape, the irregularity being sometimes increased by the extension of processes of blood into the vitreous. Occasionally a large hemispherical hæmorrhage is found at the macula, bounded superiorly by a straight horizontal line. Here the blood seems to

[1] See also "Ophth. Review," vol. vii. p. 132.

be effused between the internal limiting membrane of the retina and the hyaloid membrane, which are more loosely attached to each other in this situation than elsewhere. The blood quickly gravitates to the lower part of this space, where it is confined by the comparatively close connection between the above-mentioned membranes there existing, and we thus get a hæmorrhage of the characteristic hemispherical form. The more recent the hæmorrhage the brighter is its colour. Old hæmorrhages may be almost black. Hæmorrhages may cause permanent white spots. There may be a hæmorrhage one day, and the next a white spot in its centre. As the blood goes (which it does quickly), a white patch may remain, never so large as the hæmorrhage.

It is doubtful whether extravasations into the retina occur, however small, except from actual rupture of vessels;[1] probably the extravasations are conditioned by degeneration of minute vessels, sometimes by such capillary aneurisms as are shown in Fig. 3. White spots or brilliant plates of cholesterin are often seen in the retina adjacent to, or left by, extravasations (Pl. XI. 1). These spots, when small, may be granular; when large, they may be filmy. They are probably due to fatty degeneration of the disturbed retinal elements or of the effused blood.

Small extravasations are readily absorbed; larger ones more slowly. Sometimes pigmentary degeneration results, and an irregular black spot is left. The white spots disappear very slowly, and white granules may remain for a long time.

Symptoms.—Small hæmorrhages, away from the centre of the retina, may give rise to no symptoms. Larger ones cause loss of vision at the spot from the local damage to the retina, the loss being serious in proportion to the proximity to the macula lutea, in which a small extravasation may cause permanent loss of central vision. A ring of hæmorrhage around the macula may cause considerable central amblyopia (Pl. XI. 2). Occasionally the patient is conscious of the red colour of the extravasated blood (see

[1] According to Leber they are frequently due to diapedesis. "Graefe u. Saemisch's Handbuch," vol. v. p. 557.

under "Leucocythæmia"). At the moment of extravasation there may be no symptoms, or there may be sudden dimness of sight, or there may be ocular spectra.

Causes.—Hæmorrhage into the retina, as elsewhere, depends on one or both of two causes—increased intra-vascular pressure, decreased strength of vascular wall. Local increased blood pressure is a common cause. In optic neuritis with much constriction of the veins, the whole fundus may be covered with extravasations (Pl. VI. 1). Similar extravasations may attend all forms of retinitis. They may be large and abundant in thrombosis of the retinal vein, as Michel has shown (see p. 31). General increased blood-pressure is an occasional cause. High arterial tension may often be traced in cases of retinal hæmorrhage in which no other cause can be discovered. But it is, on the whole, a rare accident, considering the frequency with which high tension exists. Its rarity may be due to the efficient support of the retinal vessels, as explained in the description of the effects of passive congestion. It is sometimes seen when hypertrophy of the left ventricle can tell unduly on the vascular system. In the peculiar vascular condition which attends arrested menstruation, hæmorrhages occasionally occur: more rarely in suppression of some other habitual discharge. Mr. Spencer Watson[1] has recorded an instance of extensive retinal extravasation in a woman at the climacteric period, in whom there was high arterial tension, which was ultimately relieved by a copious epistaxis. Another cause is sudden loss of blood (see "Acute Anæmia").

In some cases of heart disease, especially when conjoined with degenerated vessels, numerous extravasations occur into the retina, with signs of parenchymatous retinitis, venous distension, and diffuse cloudiness. This condition has been called "hæmorrhagic retinitis." It may occur without any recognizable cardiac disease in apparently healthy persons after middle life, and is often unilateral. It probably is the result, in some cases, of thrombosis in the retinal vein. Mr. Hutchinson has adduced strong evidence to show that it is

[1] "Trans. Ophth. Society," vol. i. p. 41.

occasionally due to a gouty diathesis, acquired or inherited (see Part II., "Gout").

Degeneration of the retinal vessels is a frequent cause of hæmorrhage, although it is not often that it can be demonstrated post-mortem. It is doubtless owing to this degeneration that retinal extravasations are so common in certain general blood diseases, especially in kidney diseases and diabetes, pernicious anæmia, leucocythæmia, ague, purpura, scurvy and pyæmia, and many exhausting conditions, such as over-lactation. In some of these cases, as pyæmia and leucocythæmia, the blockade of vessels may assist. Capillary aneurisms from a case of retinal hæmorrhage in diabetes and diseased capillaries in renal retinitis are shown in Fig. 3. Jaundice is also an occasional cause of retinal hæmorrhage.

Apart from these blood diseases, retinal hæmorrhage may occur from simple senile vascular degeneration. In such cases it is sometimes produced by violent effort, such as that of a cough, or in straining during defæcation. In all conditions of vascular degeneration its occurrence is of importance, on account of its occasional association with cerebral hæmorrhage. This is well exemplified in the case of leucocythæmia (*q. v.*).

Sometimes retinal hæmorrhage results from blows upon the eye or skull. Rarely hæmorrhages are observed in young persons without discoverable cause. A remarkable series of cases in young men has been recorded by Eales, of Birmingham.[1] The only etiological condition with which it could be associated was habitual constipation. The cases will be again alluded to in the section on "Affections of the Digestive System."

The prognosis depends on the position of the hæmorrhage, and on the extent to which its causes are under control. It is worse when there are signs of general retinitis.

The chief local treatment is the application of cold and gentle pressure on the eyeball, to give temporary support to the vessels, and obtain contraction. Other measures are those suited for the general state, and for hæmorrhage elsewhere.

[1] "Birm. Med. Review," July, 1880, p. 262.

Hæmorrhage from the choroidal vessels is rare, and possesses little medical significance.

Thrombosis.—*Veins.*—Thrombosis is occasionally observed in smaller branches of the veins, which then lose their double contour—*i.e.*, their central reflection disappears, and they appear dark and large, their branches being unduly conspicuous. The condition usually depends on local causes, and has little general significance.[1]

Thrombosis may also occur in the central vein of the retina behind the eye. It is met with chiefly in the old, in whom thrombosis elsewhere is common, and has been seen in association with senile gangrene of the foot (Angelucci). But it occasionally occurs also in younger persons, in association with heart disease, aortic and mitral. Of four cases recorded by Angelucci,[2] three were in young persons, aged twenty-one, twenty-three, and twenty-four. In these it is apparently due to phlebitis. In one case[3] the vein at the spot thrombosed was thickened to three times the normal size, chiefly from changes in the external coat. The new tissue consisted of concretions such as are met with in psammomata, and was ascribed to an inflammatory process in the connective tissue of the central canal of the nerve. The thickening of the vein was so great that it must have compressed the artery. It is somewhat remarkable that the accident does not more frequently follow a primary neuritis. Only one case has been recorded in which thrombosis was supposed to have resulted from a primary inflammation.[4]

The symptoms observed have presented considerable varia-

[1] Under the title "Primary Retinal Phlebitis," Mules has lately recorded two cases where the thrombosis was confined to branches of the central vein. There was no local disease found to account for the condition, but evidence of choroiditis subsequently appeared in one of the cases. The general bearing of the thrombosis is not apparent, though Mules considers that, in one of the patients, the phlebitis was due to gout. In neither was there any optic neuritis. See "Trans. Ophth. Soc.," vol. ix. 1889, p. 130.

[2] "Ann. d'Ocul.," 1880, ii.

[3] Angelucci: "Kl. Monatsbl.," August, 1878; Zehender: "Bericht über 11 Versam. Ophth. Ges l.," p. 182.

[4] Fox and Brailey: "Ophth. Hosp. Rep.," vol. x. pt. ii., June, 1881, p. 205.

tion. There is always sudden failure of sight, often discovered on waking in the morning. It is usually incomplete, and soon presents slight improvement. In the most severe cases observed by Michel,[1] the ophthalmoscopic appearances were those of an intense hæmorrhagic retinitis. The veins were extremely distended and tortuous; the retina around the papilla was suffused with blood, beyond this zone of extravasation were circumscribed hæmorrhages, and around the macula lutea there was a greyish discoloration. The vitreous sometimes became opaque. In other cases, in which it was assumed that the occlusion of the vein was incomplete, there were merely broad striated hæmorrhages around the papilla, and round and oval hæmorrhages towards the periphery, the arteries being indistinct, and the veins dark and tortuous. In still slighter cases, supposed to be of the same nature, there were no hæmorrhages, but merely a disproportion between the arteries and the veins. In most instances the disc was little affected.

That hæmorrhages may be absent even when the occlusion of the vein is complete, is proved by the case recorded by Angelucci,[2] in which thrombosis of the retinal vein, 1 mm. behind the lamina cribrosa, was associated with senile gangrene of the foot. The veins were tortuous, but there were no hæmorrhages.

In the case recorded by Fox and Brailey glaucoma supervened, but the event is exceptional; in most recorded cases the tension of the eye was normal.

In thrombosis of the retinal vein the loss of sight is less complete than in embolism of the artery, and the ophthalmoscopic appearances differ in that the arteries, as a rule, although they may be slightly narrowed, are not empty, or filiform,—in the enormous distension of the veins,—and in the circumstance that venous pulsation can usually be observed, and that the veins may appear interrupted here and there. But in some cases the appearances simulate those of embolism very closely. There may be a cherry-red spot at the macula,

[1] "Archiv f. Ophth.," vol. xxiv. pt. 2, p. 37.

[2] "Klin. Monatsbl.," October, 1878. See also the same, January, 1880.

and in severe cases (probably in which the central artery is compressed by the distension of the vein from clot, or by the thickening of the wall which caused the thrombosis) the arteries may be extremely narrow, the veins partly emptied of blood, and the disc pale. These were the appearances in a case recorded by Angelucci,[1] in which the thrombosis was demonstrated post-mortem.

Artery.—Thrombosis has been observed in the retinal artery with ocular signs identical with those of embolism, to be described immediately. In a case recorded by Sichel it was conjoined with foci of softening and small hæmorrhages in the brain. Thrombosis in the ophthalmic artery occurs as a very rare event, and probably always as the result of thrombosis in the internal carotid. I am not aware that any case has been observed during life, but some years ago I made a necropsy on a case in which this accident had occurred. The patient, an aged man, had suffered from cerebral softening in the region supplied by the left middle cerebral artery, which was much diseased. A fortnight or three weeks before his death, there was no ocular or ophthalmoscopic change. He lay in a comatose condition, and his eyes were not again examined. Post-mortem, a recent clot was found extending down into the intra-cranial portion of the left internal carotid, fully distending it, and passing also into the commencement of the ophthalmic artery, which, however, near the eyeball, was pervious, being only partially obstructed by clot. The eyeball was quite rotten, the sclerotic of a brownish colour, and giving way before the scissors like brown paper. The retina was greatly atrophied, reduced to two-thirds of its normal thickness. Its several layers were no longer recognizable. The outer half was occupied by a thick layer of nuclei, apparently representing the two nuclear layers. Its inner half consisted of a series of lacunæ, limited by the remains of the thickened vertical fibres. No nerve-fibre layer, ganglion cells, or molecular layers could be discovered. A case of the same character, but in which a freer collateral circulation was established and the retinal changes were slighter, has been

[1] Loc. cit. 1878.

recorded by Virchow, and is described further on in the section on "Softening of the Brain." Parinaud[1] relates a case of thrombosis of the central artery of the retina, followed by symptoms of cerebral softening, in a woman aged seventy-one, who was suddenly seized with dimness of vision in the left eye, accompanied by the appearance of green and yellow spots on a grey ground. A few days later there was a central scotoma with pronounced peripheral limitation of the field of vision, and loss of colour-sense. Ophthalmoscopically the only change observed was a diminution in the calibre of both veins and arteries, followed ten days later by œdema of the retina with hæmorrhages, and capillary congestion around the macula. Three months later there was atrophy of the disc, and several branches of the central artery were filiform and white. Subsequently she developed loss of memory, aphasia, and hallucinations.

Priestley Smith[2] has urged that arterial thrombosis is the lesion in many cases that are thought to be embolism. He regards, as its causes, heart-failure (either from organic disease or other cause), spasm of the vessels or disease of their walls, and blood-states. The transient failure of sight in the opposite eye at the onset he ascribes to spasm of the retinal vessels.

Embolism.—The central artery of the retina is not unfrequently occluded by an embolus, and the occurrence is of much medical interest. Nowhere else can the phenomena of vascular occlusion be observed during life. The accident is commonly the consequence of heart disease, and is sometimes the first thing which draws attention to the existence of the cardiac affection. It was so in the case of a girl who came under my observation suffering from sudden loss of sight in one eye. On examination she was found to have a loud, distinct presystolic murmur. There was no previous history of rheumatic fever or scarlatina, and there were absolutely no symptoms pointing to cardiac disease beyond the affection of sight. Embolic infarction in other organs in many cases

[1] "Gaz. Méd. de Paris," 1882, p. 627. [2] "Ophth. Rev.," vol. iii

coexists, and the ocular accident may indicate the nature of disturbance elsewhere. It occasionally coexists with cerebral embolism, and may even furnish a warning of the probability of the latter, as in a case recorded by Landesberg, in which the ocular embolism was followed, a week later, by loss of consciousness and hemiplegia. This patient suffered at different periods from embolism of each retinal and one cerebral artery. The cerebral and ocular accidents may occur simultaneously. The diagnosis of cerebral embolism is usually sufficiently clear without it, but its occurrence is an important corroborative, and almost demonstrative, proof of the nature of the cerebral lesion. Retinal, as cerebral, embolism is rather more frequent on the left than on the right side. Its common cause is, as already stated, cardiac disease, especially mitral stenosis. It has also been observed in atheroma of the aorta and in febrile diseases, pregnancy, and Bright's disease, probably from the formation of a clot and its detachment. It may, therefore, occur at any age. A case at seventy-four years of age has been recently recorded by Hirschberg.[1]

The position of the obstruction may be in the trunk, or in one of the branches. In each case there is sudden and complete loss of sight, persistent when the obstruction is in the trunk and is permanent.[2] In rare cases the loss of sight is not instantaneous, but comes on in the course of a few minutes, commencing at the periphery. When the obstruction is in a branch, the loss of sight usually rapidly clears, except from that portion of the retina which is supplied by the occluded vessel.

The arteries beyond the obstruction are deprived of their supply of blood, and contract, so that to the ophthalmoscope they appear as fine lines only (Pl. XII. 2). They commonly,

[1] "Arch. f. Augenheilkunde," vol. v., April, 1879, p. 166.

[2] Should the retina be nourished in part by a cilio-retinal artery, embolism of the trunk of the central vessel will not cause complete loss of sight, since the retinal area corresponding to the distribution of the abnormal artery will retain its function. Such a case is recorded by Benson ("Ophth. Hosp. Rep.," vol. x., pt. iii., 1882, p. 336).

however, retain their red colour, because the contraction does not obliterate their cavity, although reducing it almost to capillary dimensions, and there is still a narrow column of blood within them. Towards the periphery, however, they are so small as to be invisible. The delicate wall of the vessel is unrecognizable, except in the larger vessels, where, on account of its contracted state, it is more distinct than normal, and appears as a white line on each side, bounding the narrow, red column. When the obstruction is complete and no collateral circulation is established, the red column may disappear, and only a white line indicate the position of the empty vessel, which gradually becomes transformed into fibrous tissue (Pl. XII. 3). In this drawing the arterial branch which passes upwards and to the left is represented only by a branching white line, while one which passes vertically upwards, and is not quite empty, is bounded on each side by a white line. Sometimes detached columns or cylinders of blood are seen in the arteries and in the veins, moving onwards in pulsatile jerks. This is probably seen only when the obstruction is incomplete.

The veins are narrowed, but less than the arteries. They are sometimes, but not always, broader towards the periphery than near the disc.

The optic disc is paler than normal, and the pallor gradually increases. The retina undergoes very marked changes, consequent on the disturbance of its nutrition. It presents a greyish or white opacity, always most marked around the macula lutea (Pl. XII. 2), and commonly also conspicuous around the disc. This opacity may come on in a few hours, but sometimes not for some days. The opacity usually stops short of the fovea centralis, leaving it of a bright red colour, so red that it was thought to be extravasation, but it is now generally believed that the tint is merely the effect of contrast with the adjacent pale opacity. The latter is believed to depend on œdema of the nerve-fibre layer, and the thinness or absence of that layer at the fovea centralis to be the cause of the usual freedom of that part from opacity (Liebreich). But occasionally the fovea may be as opaque as its vicinity, as in

Pl. XII. 2. Here I found the opacity to depend on much graver structural alterations than are usually supposed to exist. Besides evidences of œdema, there was an infiltration of all the retinal layers with lymphoid cells, similar to those of the nuclear layers, so that the thickened vertical fibres were the only structural elements which could be distinguished. The layer of rods and cones was destroyed, probably during life, in the region of the macula, because the pigment-epithelium was in contact with, and adherent to, the outer nuclear layer. In other places the thickened vertical fibres were widely separated.

Hæmorrhages are sometimes met with. The opacity commonly disappears in the course of a few weeks, but may leave

FIG. 4.—EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA (PL. XII. Longitudinal section through the artery, one-eighth of an inch (3 mm.) behind the eyeball. On each side the nerve-fibres are indicated, and between these and the vessel is much loose connective tissue. Within the contracted vessel is an oval granular mass, and in front of this is a small round body (× 300).

white spots, due to foci of degeneration. The edges of the optic disc are usually hazy. In most cases the pallor persists and increases, and passes into the whiteness of atrophy, which, at last, resembles closely simple atrophy, except in the extremely small size of the vessels.

The plug has in several cases been found after death, commonly just behind the bifurcation of the artery, in other cases in its course. In a case of embolism of the middle cerebral, and retinal artery, probably occurring simultaneously (figured in Pl. XII. 2), the artery in the nerve contained an oval granular embolus (Fig. 4). Other smaller fragments were seen in the narrowed arteries, upon the disc.

Very rarely the circulation gets re-established by the normal course. Columns of blood appear in the arteries, in part interrupted, and for a long time easily broken up by pressure. The arteries continue below normal size. Vision may be recovered, especially at the periphery, rarely at the centre. Commonly, however, obstruction remains complete.

The retinal artery is regarded as a "terminal" artery—*i.e.*, one that has no anastomoses. In most cases very little collateral circulation is set up: the arteries remain narrowed to lines as far as they can be traced. But they are visible in almost all cases as red, not as white, lines. Hence they must contain blood, persistent and therefore circulating, which has come from some slight collateral anastomoses, or from the obstruction being incomplete.

Sometimes the arteries again become pervious although diminished in size. It is probable that this is due, in some cases, to the partial restoration of the channel of the artery, and in other cases to the establishment of considerable collateral circulation. In Pl. XII. 2, for instance, the arteries are filiform only upon the disc, and as far as they remain unbranched; beyond this, they have nearly their normal size. A similar case has been recorded by Knapp. This points strongly to the establishment of a collateral circulation, probably by connection with the long ciliary arteries, although, in the researches of Leber, such connections could not, in the normal condition, be demonstrated. It is commonly supposed that the chief connection between the

retinal and ciliary vessels is by means of the vessels of the optic disc, but it is doubtful whether it is by this means that a collateral circulation takes place. The arteries are never filled in the neighbourhood of the disc, but at a distance from it. A collateral circulation in the disc may maintain the blood-supply needful to preserve the red colour of the filiform arteries, but certainly does not maintain the peripheral circulation in most of the cases in which this is re-established in a considerable degree. Probably, as Mauthner has suggested, there are, in different cases, very variable anastomoses. The re-establishment of the circulation a few hours after the obstruction, has been observed by Wood White and by Eales.[1] In each case recovery of sight occurred. It is probable that the clot either became broken up or so moved as to allow the blood to pass. In Wood White's case the event was apparently produced by pressure on the globe by the finger. The fact is of interest in connection with the occasional transient duration of the symptoms of cerebral embolism.

In Pl. XII. 3 the vessel, which is still pervious, though narrowed, is bordered for a distance by the fine white line indicating the wall thickened by contraction. The blood column within it, narrow as it is, still presents a central reflection, and towards the periphery the vessel again widens out exactly as in the other case, shown in Fig. 2 of the same plate. This broadening of the peripheral portion of the vessel nearly to its normal calibre indicates that blood enters it beyond the narrowed portion by some junction with other arteries.[2]

[1] "Ophth. Rev.," vol i., pp. 43 and 139. Mules also has recently recorded a case where plugging of a branch of a retinal artery disappeared, under massage of the globe, about an hour after its occurrence. The visual field was restored forthwith, with the exception of a small area corresponding to the immediate neighbourhood of the embolus. ("Trans. Ophth. Soc.," vol. viii., 1888, p. 151).

[2] It is greatly to be desired that, in any post-mortem examination of a case in which there has been embolism of the retinal artery, and in which such collateral circulation is established, a ligature should be placed around the artery, or around the optic nerve in front of the entrance of the artery, and the ophthalmic artery then injected, so as to discover the channels by which the circulation is established, and which elude observation under normal conditions.

In partial embolism the segment of the retina, to which the occluded branch goes, becomes opaque, and is sometimes the seat of numerous hæmorrhages. Both opacity and extravasations ultimately disappear. The corresponding portion of the optic disc may be normal, as in the case shown in the figure, or it may be atrophied. In one case on record it was hyperæmic (De Wecker). The corresponding vein is at first distended, afterwards smaller than normal.

Embolism of the trunk of the central artery commonly causes complete and persistent loss of sight. When the occlusion is of a single branch, there may be a complete initial loss of sight, due probably to the plug causing a temporary obstruction in the trunk of the artery before it passed on to the branch in which it was arrested. Occasionally, in such cases, the blindness has remained complete, although the ophthalmoscope afterwards demonstrated that only one branch of the artery was occluded. The general retinal anæmia may, in such cases, have been so prolonged that the nerve elements suffered a shock, damaging their nutrition beyond the power of recovery on the succeeding restoration of the circulation. Commonly, in such cases, the permanent loss

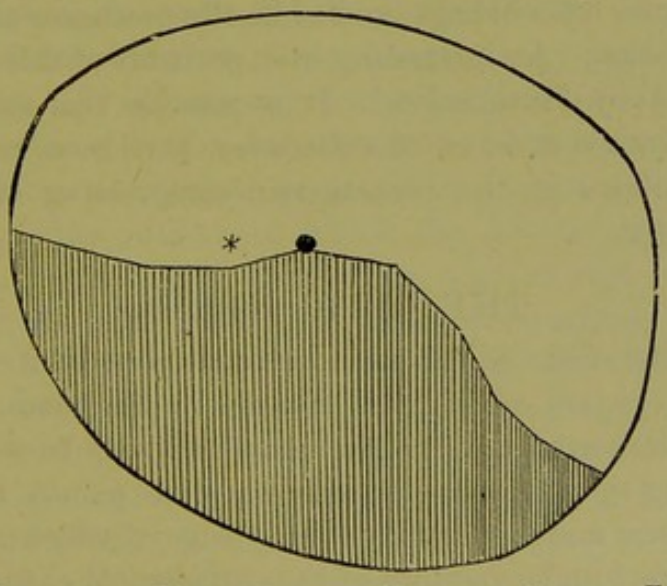


FIG. 5.—DIAGRAM OF RIGHT FIELD OF VISION IN PARTIAL EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.

The descending branches of the central artery were normal, but those proceeding upwards were empty. The shaded area indicates the portion of the field in which sight was lost. The asterisk indicates the position of the fixing point, the dot that of the blind spot.

is of a portion of the field corresponding to the distribution of the branch plugged. When this is one of two primary divisions of the artery, the loss may amount to one-half of the field; when of a smaller branch, to a quadrant, or the like. There was a loss of nearly one-half in the case figured in Pl. XII. 2, in which one branch running upwards and outwards is completely obliterated, and others running upwards and inwards are partially obliterated. The loss was that shown in the adjacent diagram of the field of vision (Fig. 5).

Occasionally, sudden blindness has occurred, and the retinal arteries have appeared narrow, recovering their normal size after a short time, with restoration of vision, as in the cases of Wood White and Eales, discussed above.

Arterial ischæmia, similar to that which results from embolism, has been ascribed to a retro-ocular hæmorrhage around the artery compressing it. The ophthalmoscopic distinction of this from embolism is uncertain, and probably depends rather on the incompleteness of the ultimate obstruction than on any differences in the early retinal appearances. It is said to occur in cases in which there is a general tendency to hæmorrhage, and to be the precursor of cerebral extravasation. An interesting case, probably of this character, is related by Hutchinson.[1] It is possible that some of the cases supposed to be of this character have been really cases of thrombosis in the central vein compressing the artery (see p. 30).

THE OPTIC NERVE.

The alterations in the optic nerve, as seen at its entrance into the eye, are among the changes in the fundus oculi of greatest importance to the physician. It may be well, before describing those changes, to consider some points, regarding its structure and appearance, a knowledge of which is essential for a correct understanding of the pathological changes.

In the optic disc we have presented to view the termination of a nerve—a structure consisting of nerve fibres, a little supporting connective tissue (especially abundant around the

[1] "Ophth. Hosp. Rep.," October, 1874, p. 51.

central vessels), and a number of blood-vessels, for the most part capillaries, which confer on the disc its tint. The nerve fibres radiate and spread out in the retina, but not equally on all sides, being few on the temporal side, towards the macula lutea, and numerous on the nasal side and especially above and below. The minute vessels of the disc are derived partly from the posterior ciliary (choroidal) arteries, and partly from the central retinal artery, twigs from both of which commonly unite in forming the "circle of Haller," a series of vessels which surround the optic nerve behind the disc. The connective tissue between the bundles of nerve fibres is small in quantity, but contains scattered nuclei. The opening in the sclerotic is funnel-shaped, the wider part being posterior. The termination of the nerve fits pretty closely into the inner, smaller, part of the opening, while the space between the nerve and its outer sheath, "vaginal space," passes up into the posterior part of the opening (Fig. 16).

The separation of the optic nerve fibres to radiate into the retina leaves the central hollow known as the "physiological cup," the size and depth of which are determined by the arrangement of the nerve fibres. The vessels are chiefly developed among the nerve fibres and towards the surface of the disc, and hence the central cup is always much paler than the periphery. It is commonly white, but sometimes mottled grey from the reflection of the white trabeculæ of the "lamina cribrosa," which closes in the sclerotic foramen, and through the meshes of which the greyer, now non-medullated, nerve fibres pass. The tint of the circumferential portion of the disc is, as already explained, deepest where the nerve fibres are most numerous, and hence the nasal half of the disc is naturally redder than the temporal half. The arrangement of the nerve fibres also causes the side of the central cup to be steep on the nasal and shallow on the temporal side, the difference being proportioned to the inequality with which the nerve fibres are distributed. When the fibres are almost all packed on the nasal side, the cup may be very large, and extend on the temporal side to the margin of the disc. Often, however, there is no paler central cup.

The boundary of the "disc," as commonly recognized, is the choroidal ring, *i.e.*, the edge of the opening in the choroid corresponding to that in the sclerotic. The latter is usually the smaller of the two, and hence a narrow rim of sclerotic commonly appears within the choroidal edge, and is known as the "sclerotic ring." It is often visible only on one side. At the passage of the nerve fibres over the edge of the sclerotic, they curve a little above the level of the retina, and this slight prominence has suggested the name of "optic papilla" as a designation for the area of entrance of the optic nerve.

The trunk of the optic nerve possesses a double sheath: the inner is delicate, closely invests the nerve, and is continuous with the pia mater of the brain. The outer sheath is thicker and fibrous, blends in front with the sclerotic, and is continuous at the optic foramen with the dura mater. There is not, as was once thought, a reflection of the arachnoid at the optic foramen, and thus the vaginal space of the optic nerve—*i.e.*, that within the outer sheath—is continuous with the subarachnoid and subdural spaces around the brain. This vaginal space is traversed by tracts of tissue connecting the two sheaths. At the anterior extremity of the nerve, the space passes within the posterior part of the sclerotic opening, and is, according to some authorities, closed; but, according to others, it is continuous with lymphatic spaces in the substance of the optic nerve, and probably also in the retina.

The optic nerve, at its entrance into the eye, undergoes certain pathological changes in common with the retina. When the retina is generally inflamed, and when it is atrophied, the optic "papilla" participates in the change. But it also undergoes inflammatory changes independently of the retina.

The pathological conditions of the papilla resolve themselves, from their clinical features, into two groups,—increased vascularity, commonly with increased prominence; diminished vascularity, commonly with shrinking. The states characterized by the former are more or less inflammatory, and are often included under the generic term

"optic neuritis." Those characterized by the latter signs are accompanied by wasting of the nerve tissues, and are included under the generic term "optic nerve atrophy."

It must be remembered that the term "optic nerve" is employed in two senses—to designate the whole nerve, and also its intra-ocular termination as seen with the ophthalmoscope. To prevent the confusion arising from this double use of the words, it has been proposed by Leber to use the terms "optic nerve" and "optic neuritis" when speaking of the whole nerve, employing only the words "papilla" and "papillitis" to designate the intra-ocular termination of the nerve and its inflammation. This distinction has not, however, come into general use in this country. The custom of employing the term "optic neuritis" as a designation for the intra-ocular inflammation, makes it inconvenient to restrict it to inflammation of the nerve trunk, but for the former condition the synonym "papillitis" is very useful.

The characters of these morbid states may be thus tabulated:—

A.—Morbid states of the optic nerve, characterized by increased vascularity or signs of inflammation.

1. Simple congestion of the disc; undue vascularity, redness, softening but no obscuration of the edge, and no swelling.
2. Congestion with œdema of the disc (slight neuritis or papillitis); increased redness, with slight swelling; obscuration of the edge of the disc, complete to the direct examination, incomplete to indirect examination.
3. Neuritis, or papillitis; increased redness and swelling, with obscuration of the edge of the disc, complete in degree, partial or total in extent.

B.—Diminished vascularity and signs of wasting.

1. Simple atrophy; increased pallor from the first; "primary atrophy."
2. Congestive atrophy; secondary to congestion; pallor slowly succeeding simple congestion.

3. Neuritic atrophy, succeeding pronounced neuritis; "consecutive atrophy," "papillitic atrophy."
4. Atrophy succeeding choroiditis and retinitis; "choroiditic" and "retinitic atrophy."

A.—*MORBID STATES CHARACTERIZED BY INCREASED VASCULARITY OF THE DISC, OFTEN WITH SIGNS OF INFLAMMATION.*

Simple Congestion.

Increased redness is the universal expression of tissue hyperæmia. But it is of less value as a sign of hyperæmia of the optic disc than in the case of most tissues, on account of the great variation in the amount of natural redness. Attention to tint of disc alone is a prolific source of error in ophthalmoscopy. It is as if a small portion in the centre of the cheek were examined to determine whether or not there was pathological hyperæmia. Nevertheless, abnormal redness of the disc does occur as a morbid state, and, although in itself a sign of little value, it derives importance from certain concomitant conditions. It is significant (*a*) when it possesses special characters to be immediately described; (*b*) when developed under observation; and (*c*) when it is notably greater in one eye than in the other: but even in the latter case there is room for error in the possibility of a natural difference, or that the paler eye may present an abnormal decrease in redness.

There are certain characters which aid very much in the recognition of the pathological increased vascularity. First, morbid redness has usually a tendency to invade the physiological cup, and often, especially when the cup is small and shallow, to obscure it altogether. Secondly, pathological redness has a tendency to render the sclerotic ring or the edge of the choroid indistinct; to blur the sharpness of the outline of the disc. The change, when *very slight*, may be best appreciated by examination with the indirect image (Pl. I. 1). It is due to the circumstance that the seat of the vascularity is the layer of nerve fibres, and it may extend in

front of the edge of the disc; and it is often attended with a little swelling of the nerve fibres or effusion of fluid, which conceal the structures beyond. The term "congestion" is, I think, best restricted to those cases in which the increased vascularity exists alone, with so little structural change, that the edge of the disc can still be perceived by both methods of examination, although not so sharp as natural.

The redness is different from the ordinary tint of the discs. It is brighter, softer, somewhat velvety in aspect, sometimes finely stippled. Occasionally, however, the tint of a disc thus changed may be positively paler than normal, although the uniformity of the colour, and the softened edge, afford evidence of the pathological character of the change on which it depends. This paler tint is often seen when the condition is passing into atrophy. The retinal vessels are usually unchanged; their walls are often conspicuous, by contrast with the redness of the disc, as white lines bounding the blood column, and any white connective tissue which surrounds them at the point of emergence is also unduly conspicuous (Pl. I. 2). The appearance is suggestive of the white tissue being a pathological result of the hyperæmia. It is probable that it is so, but the appearance is too common as a physiological condition to have much value. Its distinctness possesses a little significance, as due to the invasion of the middle of the disc by the hyperæmia. Occasionally, when the hyperæmia of the disc is the expression of graver changes behind the eye, the arteries may be narrowed in consequence of retro-ocular pressure.

The condition thus described as "simple congestion" of the disc is usually a chronic state, and corresponds, probably, to the condition which has been called by Clifford Allbutt "chronic neuritis." The evidence that there is actual inflammation does not seem sufficient to warrant the application to this state of the term "neuritis," especially as there are forms of true neuritis characterized by extreme chronicity. It is rare, I think, that such simple hyperæmia is the first stage of an actual neuritis. In the latter, swelling comes on *pari passu* with the hyperæmia—*i.e.*, congestion

with œdema, rather than simple congestion, is the first stage of neuritis. The simple congestion is occasionally seen as a substantive condition, and may, in rare cases, precede atrophy. The condition may be the expression of a state of congestion and degeneration in the whole optic nerve, or be apparently limited to the optic disc. It is not unusual in cases of hypermetropia. It may occur as a consequence of injuries, blows, &c., in the neighbourhood of the eye. The affection of sight which results from the use of tobacco may be attended with this state. It occurs also, probably, from other toxic agents, as lead. It has been observed by Clifford Allbutt in general paralysis of the insane; and, although some other observers have failed to find it, one case under my observation presented it very distinctly. It has also been described by the same writer as accompanying atrophy in locomotor ataxy; but in that disease, although often looked for, it has not been found by others or by myself. It is sometimes present in cases in which there is reason to believe a similar condition exists in the brain. The figures Pl. I. 1 and 2 are from a patient with cerebral embolism, in whom the condition came on in association with secondary brain irritation—mental failure and rapid wasting in the paralyzed limbs. I have seen a similar appearance in acute mania. Microscopical examinations of the disc in this state are rare. Clifford Allbutt examined one case and found only distension of the minute vessels with that granular degeneration of the nerve fibres present in all forms of atrophy.

Optic Neuritis or Papillitis.

Congestion with Œdema (Pl. I. 3 and 4) is really the first stage of papillitis. The normal rosy tint of the disc becomes increased; its edge is blurred, but is recognizable on indirect examination. There is a pale reflection from the adjacent retina, surrounding the disc with an indistinct halo (Plate I. 3). On examining the disc by the direct method, the morbid appearance is much more marked (Plate I. 4). The edge of the disc is lost, and the opacity is seen to be in part the result of an undue distinctness of the

radiating striation of the nerve fibres as they course on to the retina. It must be remembered that this striation is often visible as a normal condition, especially above and below. Where the aggregation of the fibres is very close, the central cup being of large size, the appearance of commencing œdema may be closely simulated. In the latter condition, however, there is from the first more or less invasion of the central cup, which soon becomes obscured. The increased vascularity of the disc may be striated at the periphery. There is often distinct swelling. The centre of the papilla may be, as in the figures referred to, much redder than the periphery, on account of the slighter central swelling allowing the vascularity of the disc to be perceived. In the periphery the tint of the choroid is concealed. The retinal vessels may be normal, or the veins may be enlarged.

It is important to note that the direct examination renders these changes more distinct. If the obscuration of the edge of the disc is apparent only, in consequence of the similarity in tint of the disc and the adjacent choroid, the edge of the disc is more distinct on direct than on indirect examination. When the indistinctness of the edge is due to the opacity of the structures in front of it (except in the very slightest form), the edge is less distinct on direct than on indirect examination. This is no doubt due mainly to the fact that the illumination is stronger and the plane of focus is less exact in the indirect method, so that the choroidal edge and the tissue in front of it are in view at the same time; whereas the direct method of examination, by its higher magnification and more exact focussing, shows the tissues in front of the edge so as to increase the concealment of the latter. This fact will often be found of service in distinguishing between a normal redness and an abnormal obscuration of the disc. Of course, it will not distinguish the latter from the cases just mentioned, in which there is a slight physiological obscuration of the edge by nerve fibres.

This condition of œdema is usually an acute affection, and is commonly the first stage of neuritis. It is said to be an effect of pressure on the retinal vein, causing passive conges-

tion of the retinal vessels, and it has been described as the result of the general passive congestion of heart disease. In these cases it may be associated with retinal hæmorrhages. It may, however, occur as the first stage of neuritis without the least sign of mechanical congestion. In the case figured, there was probably a syphilitic intra-cranial node or growth.

Neuritis (Papillitis).[1]—From congestion with œdema to actual inflammation the transition is one of degree. It seems better to restrict the term neuritis, or papillitis, to those cases in which the swelling and opacity are sufficient to conceal the edge of the disc both on direct and indirect examination. This condition is found in most cases to result not merely from vascular congestion and œdema, but from changes in the nerve fibres and connective tissue, such as we regard as evidences of inflammation. The change may completely veil the whole or only half the disc; and from such a slight degree of neuritis to the most intense form we may have every gradation, characterized by very considerable differences in appearance.

These variations in the appearance of the disc in different cases, and supposed differences in their origin, have led to the establishment of two varieties of the affection, "descending neuritis," and the "choked disc," "Stauungs-papille." The grounds on which these divisions have been made are, as will be shown, uncertain, and it is better in the first place to consider the common features which all forms of papillitis possess. The supposed varieties and theories of this origin will be then better understood.

A case of optic papillitis of considerable intensity presents, in the course of its development, certain stages, the general features of which are usually recognizable. The transition from one to the other is, of course, a gradual one, and cases are seen which present appearances intermediate between the several stages. Moreover, at any stage, the morbid process may stop, remain stationary for a time, and then recede. This may occur spontaneously or as the result of treatment.

[1] Plates I. 5, 6, II. 1, III., IV., V., VI., VII.

Thus certain *forms* of neuritis may be distinguished according to the intensity of the changes, but our knowledge of the conditions on which they depend seems insufficient at present to distinguish them as varieties otherwise than as varieties of intensity, on whatever differences of mechanism they may ultimately be proved to depend.

The first stage of optic neuritis is that which has been already described as "congestion with œdema"—a condition of increased redness, swelling, and cloudiness, masking the edge of the disc to direct, but leaving it perceptible to indirect, examination. In this condition the microscope reveals no sign of tissue inflammation. The second stage, that of actual, but slight, neuritis, is characterized by the disappearance of the edge of the disc even to indirect examination (Pl. I. 5). The transition from the disc to the retina is gradual, the edge is "blurred," and its position has to be guessed at. When this is the case, the disc always, I believe, presents not only œdema but also changes in its tissue elements which indicate a process of inflammation—proliferation of nuclei, escape of leucocytes, and degeneration of nerve structures—changes similar to those which are regarded in all organs as indicative of inflammation.

The red tint of the disc becomes more marked, so that it may be almost the same as that of the adjacent choroid, or it assumes a reddish grey tint, which is very characteristic, and the disc loses its normal semi-translucent appearance. The swelling increases, and is easily recognized, even on indirect examination, by the relative displacement of different parts on lateral or vertical movement of the lens. The striation of the periphery, perceptible in the first stage, increases, but becomes redder. It is due not only to the swelling and opacity of the nerve fibres, but also to the minute vessels which course between them. In the centre of the disc the redness is stippled or uniform, not striated, and the centre is commonly distinctly darker red than the peripheral portion (Pl. I. 6). The centre may be red, and the periphery greyish red. The striated edge passes, by gradation, into the tint of the adjacent fundus. The physiological

cup often disappears during the stage of œdema; if large, a trace of it may remain to the stage of commencing neuritis, but is rapidly encroached upon and covered in by the swelling of the papilla (see Pl. III. 2, in which it has almost disappeared).

White lines and spots are not uncommon, especially in the cases in which the changes remain of slight degree. They often correspond to the position of arteries (Pl. III. 3). The swelling and obscuration may involve all parts of the disc equally, especially in the more acute forms of neuritis, or it may be much more marked on the nasal than on the temporal side of the disc. The difference may be so great that the position of the edge of the disc may be distinct on the temporal side, while the nasal edge is completely obscured by opaque tissue—a condition which may for brevity be termed "hemi-neuritis" (Pl. V. 1, 2, 4). Hæmorrhages are not uncommon in this stage, sometimes on the surface of the swelling, or even on the least changed part of the disc (Pl. V. 1) or just beyond its edge (Pl. V. 4). They are always small. The arteries usually present little change in the slighter stage of neuritis, although often recognized with difficulty on account of the colour of their blood corresponding to the tint of the disc. They are a little concealed at their emergence, but have a nearly straight course. Arterial pulsation has been observed by Graefe[1] and Becker.[2] The veins lose their central reflection as they pass down the sides of the swelling, and appear dark. They may or may not present dilatation, indicative of mechanical congestion. In the early stage of papillitis from tumour, as a rule, they less frequently present dilatation than in that from meningitis.

As the papillitis goes on, the swelling increases, and becomes often so great, that there may be a difficulty in seeing the surface of the swelling by the direct method without the use of a convex lens. The veins, as they curve down the sides of the swelling, appear still darker and

[1] "Arch. f. Ophth.," xi. pt. 1, 201, and xii. pt. 2, 131.
[2] "Wien. Med. Wochenschrift," 1873, p. 34.

foreshortened, and are concealed, just beyond its edge, in the adjacent retina (Pl. III. 4, IV. 1, V. 6). The veins commonly now present some enlargement, often considerable, and the arteries are narrowed. They may be indistinct upon the disc, being concealed by the tissue. The arteries are always more concealed than the veins. The vessels are often lost to view at the centre of the swelling (Pl. I. 6, IV. 3, V. 5), although there may be a depression where they emerge. This central depression is sometimes large, in consequence of the neuritic swelling being chiefly located on the edge of the disc (Pl. VII. 1)—a condition which has been distinguished as "perineuritis." The swelling increases, not only in height but in lateral extent, and partly displaces, partly invades, the adjacent part of the retina, often having a diameter two or three times that of the optic disc. There are, however, rarely signs of any general disturbance of the retina. Extravasations of blood may occur on the surface of the swelling, and not uncommonly white, flake-like spots may appear upon it, often concealing the vessels (Pl. III. 3, IV. 1, VI. 2). Occasionally a white spot is surrounded by a halo of hæmorrhage (Pl. VI. 2). Sometimes similar spots exist in the retina close to the edge of the disc.

A large number of cases proceed no farther than this stage. Signs of passive congestion of the veins may or may not be present. If not present before, they may be developed during the subsidence of the neuritis, especially if quick absorption of the inflammatory products cannot be obtained. Neuritis of this stage may clear completely (Pl. IV.), the inflammatory products being for the most part removed, and those which remain merely causing a little increase of tissue in the middle of the disc. The subsidence is marked by a diminution in the height and extent of swelling, and in its redness. At first it may appear somewhat more opaque (Pl. VI. 3), but becomes less so as the swelling subsides. The position of the edge of the choroid becomes appreciable, and gradually clearer, first on the temporal, and then on the nasal side. The disc has a "filled in" aspect (Pl. VI. 5), and both arteries and veins may be narrowed and partly

concealed on its surface. This is especially the case when the new tissue-elements in the disc have been sufficiently abundant to develop signs of strangulation during the inflammatory stage (Pl. VI. 1 and VIII. 1). When this is not the case, as in Pl. III. 5 and 6, IV. 1 and 2, the disc may rapidly clear in the centre, as well as in the periphery, and the physiological cup be quickly reproduced. Often, however, white lines along the vessels indicate the remnants of preceding inflammation (Pl. IV. 2, II. 4), and the vessels may be a little narrowed. Commonly, when the inflammatory swelling has been marked, a disturbance of the pigment-epithelium leads to a narrow zone of atrophy adjacent to the disc (Pl. II. 4, IV. 4).

Whether or not there are signs of mechanical congestion in the stage of neuritis just described, a further increase in the inflammation is invariably accompanied with signs of compression of the vessels, and strangulation of the inflamed papilla, with a rapid and intense increase in the mischief. The tumour formed by the swollen papilla becomes much more promiment, and extends laterally in all directions, even as far on the temporal side as the macula lutea. The form of the swelling varies; sometimes it remains conical, but usually the sides become steeper, and the top more or less flattened. The sides may even overhang so that the tumour has a fungiform shape, and the vessels, as they pass over the side, may be concealed by the edge of the swelling, and reappear in the fundus in a different position. Good examples of this intense strangulated neuritis are represented on the next page (Figs. 6 and 7, and at Pl. VI. 1).

The arteries are much narrowed, and often altogether invisible on the swelling, being buried in its substance, and appearing first in the retina, a little distance from its edge. The veins are often concealed on the disc, at least in part, but some of them are usually visible towards the edge of the swelling, and are greatly distended. When the amount of swelling is extreme, all the vessels may be concealed, as in Pl. VI. 1. Hæmorrhages are frequent and extensive, and are commonly situated on the edge rather than on the

surface of the swelling (Pl. VI. 1 and VIII. 1). The overhanging edge may be infiltrated with blood. The veins may

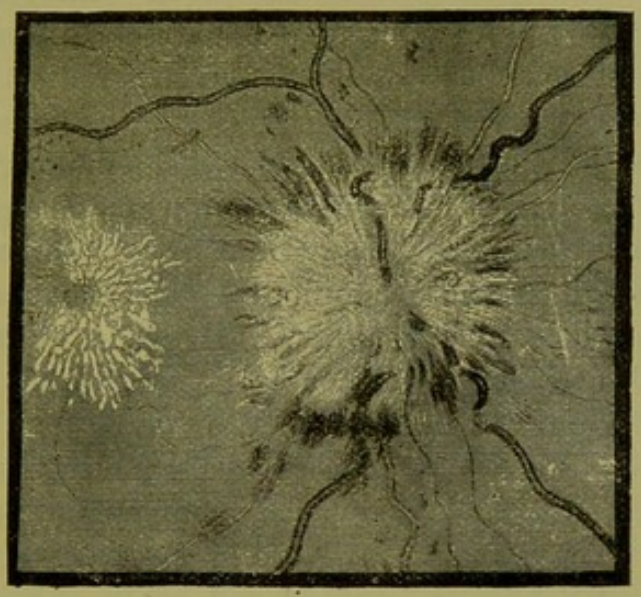


FIG. 6.—ACUTE OPTIC NEURITIS IN A CASE OF CEREBRAL TUMOUR.[1]

There are great swelling of the disc, which is surrounded by radiating hæmorrhages, and, at the macula, a star-like arrangement of white spots. No albuminuria, and no history of syphilis.

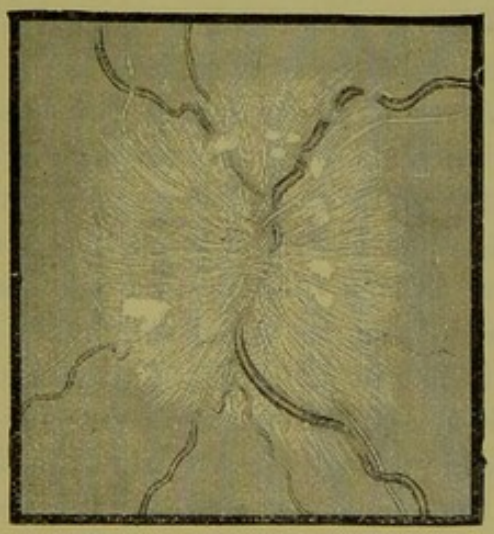


FIG. 7.—ACUTE OPTIC NEURITIS.

The veins and arteries are both concealed by the swelling. The veins are distended, while the arteries are narrowed. Numerous white patches are scattered over the swollen papilla.

[1] After Edmunds, "Trans. Ophth. Soc.," 1884, p. 291.

be concealed beyond the edge of the swelling, and often present many curves and twists, sometimes corkscrew-like from their elongation. The tint of the strangled swelling is usually a full red, mottled and streaked from enlarged vessels and small extravasations. The striation due to the nerve fibres is commonly lost. The retina adjacent is often the seat of hæmorrhages, which may extend along the vessels from the disc. Not unfrequently secondary changes occur in the retina over a wide extent. Hæmorrhages, usually striated and situated in the nerve-fibre layer, may be scattered over the whole fundus (Pl. VI. 1). The veins are often distended, and may be tortuous for a long distance from the disc. The ultimate distension of the veins may be as great in the papillitis which succeeds a descending neuritis (see Figs. 18, 23, &c.) as in that which is supposed to be limited to the eye. The retina, in rare cases, presents areas of opacity, diffuse and cloudy, or localized and white, and often occurring along the course of the vessels (Pl. VIII. 1). When the swelling of the retina is very intense it may become thrown into folds. On examining such an eye, bright streaks will be seen running in different directions, frequently arranged radially around the macula, probably due to the reflection of the light from the summit of the folds.

The time taken for the development of these changes varies within wide limits. A neuritis may remain for months and even years in the slighter degree, or most intense strangulation may be developed in a few weeks.

Subsidence of Neuritis.—The gradual subsidence of the slighter degrees of neuritis has been already traced. In the more intense forms, in which strangulation has occurred, the stage of subsidence presents certain peculiar features. The venous distension gradually lessens after the strangulation has existed for a time, and the veins may become narrow before other signs of strangulation subside. In Pl. VI. 1 they are much smaller than in the earlier stage of strangulation shown in Pl. VIII. 1. This is probably because the compression of the arteries becomes sufficient to lessen the blood-supply to such an extent as to permit the veins to recover nearly their

normal calibre. When the strangulation is less intense, the commencement of subsidence may be attended with an increase in the mechanical distension of the veins, and increased narrowing of the arteries. The redness of the swelling lessens, hæmorrhages, as a rule, cease to occur,[1] and some of the blood already extravasated disappears. The tumour lessens in height and in extent, and, if fungiform, again becomes conical (Pl. VI. 3). The highest portions of the swelling gradually become pale; the sloping sides and adjacent part of the retina may present a darkish discoloration, into which the central pallor passes gradually (Pl. II. 1, IV. 5, V. 6, VI. 3). The centre of the swelling soon presents a distinct depression, from which the vessels emerge often concealed by whitish tissue. Over the swelling the course of the veins becomes more distinct. The arteries may be still concealed, their more rigid, straight course having caused them to be buried in the new tissue more deeply than the veins, which were pushed up before it, and the paler tint of the arteries also renders them less conspicuous. The concealment of the veins beyond the edge of the disc is even greater than it was before, in consequence of the curve of the inelastic vessels into the retina being increased as the swelling subsides (Pl. VI. 4 and 5). Slowly the pallor increases and the swelling lessens, although the constriction of the vessels may increase, in consequence of the cicatricial contraction of the newly-formed tissue. Occasionally, when large vessels appear on the papilla during the neuritis, these become tortuous, and gradually disappear during subsidence (Pl. IV. 5). As the white area narrows to near the limits of the disc, the edge of the choroid and sclerotic appear, dimly at first (Pl. VI. 4, left edge), then more distinctly. The disc has a white "filled-in" look (Pl. VI. 5, VIII. 2), the vessels are constricted, and it is very long before any central depression is developed on the disc, although ultimately the contraction of the fibrous tissue, as in other cicatrices, proceeds to an extreme degree, and the

[1] Very rarely fresh hæmorrhages form adjacent to the papilla during the stage of subsidence, as in Pl. VI. 4.

disc may again become hollow (Pl. IV. 6). The lamina cribrosa is, however, usually permanently veiled, an important characteristic of this form of atrophy. The retinal pigment and choroid are frequently disturbed near the disc, and a zone of irregular pigmentation with slight choroidal atrophy is left, causing the disc to have irregular edges, but this zone is not always proportioned to the amount of inflammatory disturbance, and if slight the disc may ultimately come to have a clean-cut edge. At first the disc is usually very white, rarely grey, with white lines along the vessels (Pl. II. 2, upper half). When it has reached the retinal level, however, although it may appear white to the indirect image, a faint grey tint is usually perceptible on direct examination, and as the contraction increases this grey tint becomes more marked, and the ultimate appearance of the disc is usually distinctly grey to direct examination, although often white to indirect examination. Very rarely the inflammation may subside irregularly, clearing from one part of the disc, while the other still presents the characters of neuritis (Pl. II. 2).

The retina undergoes certain changes during this period of subsidence. Hæmorrhages upon it are usually soon absorbed, but sometimes undergo transformation into spots of pigment. Some extravasations lead to the formation of white spots in the retina. This is especially the case near the disc, where the nutrition of the retina is always a good deal disturbed by the adjacent inflammation. These white spots, which depend on fatty degeneration, either of fibrin or of the retinal elements, and persist after the blood has been removed, commonly originate close to the borders of the neuritic swelling; but as the latter subsides and contracts, they are left behind, and are often one or two discs' breadth from the edge of the sclerotic ring, and they may then puzzle the observer from their resemblance in character and position to the spots of albuminuric retinitis. A group of such spots, midway between the disc and macula lutea, is seen in Pl. VI. 3. The degenerative changes which occur when the inflammation is very intense, and of wide extent, may leave an appearance

strikingly similar to that of the albuminuric affection. If the swelling approaches the macula, degeneration occurs among the radiating fibres of the fovea centralis, causing spots identical in appearance, and probably in nature, with those which in renal disease form the familiar stellate figure around the macula. A striking instance of this is shown in Pl. VIII. 2. The distinction, as will be subsequently explained, consists mainly in the evidence the disc affords of a considerable antecedent neuritis. Frequently, as the retina becomes atrophied, slight pigmentary deposit takes place in it, especially around the macula lutea, and sometimes the atrophy is accompanied by wide-spread slight disturbance of the pigment-epithelium.

When a neuritis has lasted a long time, and the veins have been persistently stretched over the swelling, they may be so permanently elongated that the subsidence of the neuritis, instead of being attended with a diminution in their tortuosity, is accompanied by an increase in their curves. This is shown in Pl. VI. 4 and 5, in which also a very rare circumstance is presented—the occurrence of recent extensive hæmorrhages during the stage of subsidence.

Second Attacks of Neuritis.—If a disc has become completely atrophied it is very rarely again the seat of inflammation. In one case, however, of a boy aged twelve (under the care of Dr. Hughlings-Jackson), who had double optic atrophy, and absolute blindness due to intra-cranial disease some years previously, distinct double papillitis occurred in the atrophied discs, associated with symptoms of intra-cranial tumour. When, however, atrophy is partial or absent, in rare cases two attacks of neuritis may occur. In one case, for instance, a patient suffered without doubt from a cerebral tubercle, and died from an attack of tubercular meningitis. The former had probably become quiescent, and the neuritis which it caused subsided, leaving partial atrophy. The discs again became swollen and obscured with the symptoms of meningitis.

Pathological Anatomy.—In the condition described as

"congestion with œdema," the microscope reveals less prominence than was observed during life, because the swelling

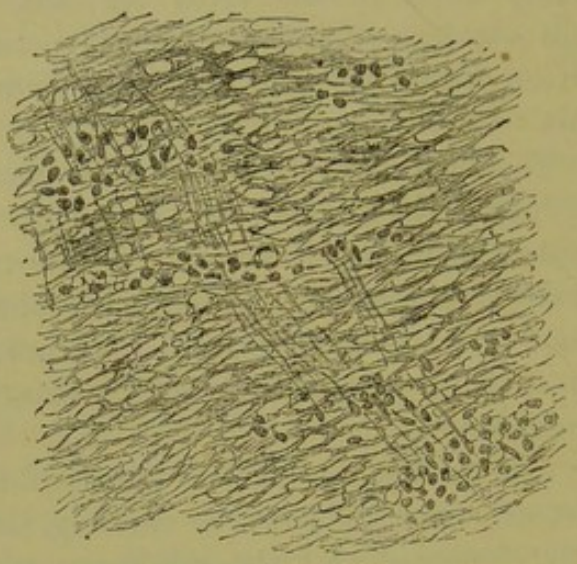


FIG. 8.—OPTIC NEURITIS; NERVE-FIBRE LAYER.

The fibres are separated by numerous round and oval spaces, due to œdema. The nuclei are unduly numerous, and lie in groups, which indicate the fasciculi. (× 150.)

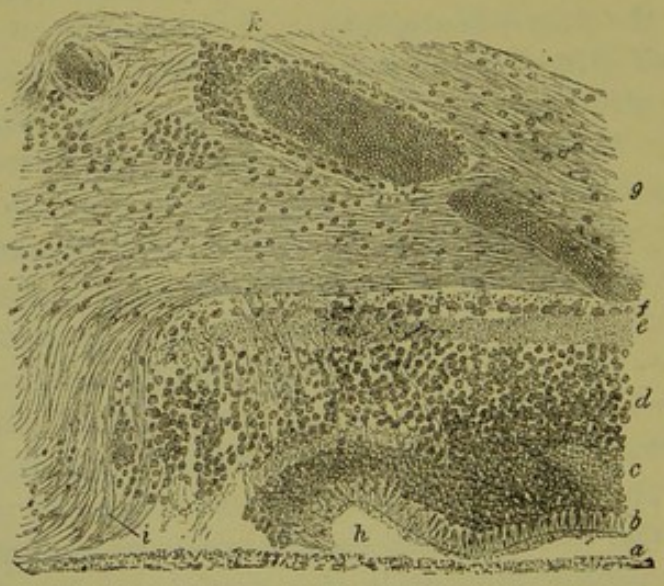


FIG. 9.—SECTION THROUGH THE OUTER PART OF AN INFLAMED PAPILLA.

(*a*) Pigment-epithelium. (*b*) Layer of rods and cones. (*c*, *d*) The nuclear layer. (*e*) The inner molecular layer. (*f*) Ganglionic cell layer. (*g*) The greatly swollen nerve-fibre layer, containing many leucocytes, many of them surrounding the vessels. (× 150.)

depended on distended vessels and effused serum. The nerve fibres are separable with abnormal readiness, and are divided by spaces which during life were occupied by serum (Fig. 8). The fibres themselves may present slight varicosity. There is no increase in the connective-tissue elements, and there are no products of degeneration of the nerve fibres. The retina is normal to the edge of the choroid, its nerve-fibre layer being alone increased in thickness by the conditions mentioned as causing the swelling of the papilla. Sometimes the retinal layers may be displaced outwards a short distance.

In the stage of developed neuritis (Figs. 16—21, &c.), the microscope reveals a considerable swelling of the papilla, often two or three millimetres above the level of the

Fig. 10.—Section through an Artery and Vein in the same Papilla.

(*a*) Distended vein; (*b*) contracted artery with thick walls.

Fig. 11.—Optic Neuritis; Collection of Leucocytes in a Perivascular Space. (× 150.)

Fig. 12.—Section through an Inflamed Papilla.

The vessels are distended with corpuscles, and several of them surrounded by leucocytes. The nerve fibres, separated by œdema-spaces, course upward and to the left, and at right angles to them are seen some fine connective-tissue (supporting) fibres. (× 120.)

choroid. There is usually a central depression, which may be larger and deeper than the ophthalmoscopic examination suggested. The swelling may be very distinct to naked-eye examination (Figs. 27, 28, p. 65), and hæmorrhages may be seen upon it. Thus, mere inspection of the fundus after removal may show the previous existence of papillitis. The swelling is due to several conditions, the relative degree of which varies much in different cases:—(1) The vessels, large and small, are distended with blood (Fig. 12). (2) Spaces between the nerve fibres sometimes indicate the persistence of œdema (Fig. 8). (3) Many nuclei are seen, some of which are leucocyte-like corpuscles, most abundant around the vessels, which may be encrusted by a thick layer (Figs. 9*k*, 11, 12*a*, &c.); they are sometimes grouped into dense masses

Fig. 13.—Granule-Corpuscles, &c.

From the substance of the papilla in a case of optic neuritis. (Glycerine preparation; × 100.)

Fig. 14.—Varicose Nerve Fibres

From an inflamed papilla in a case of tubercular meningitis. (× 200.)

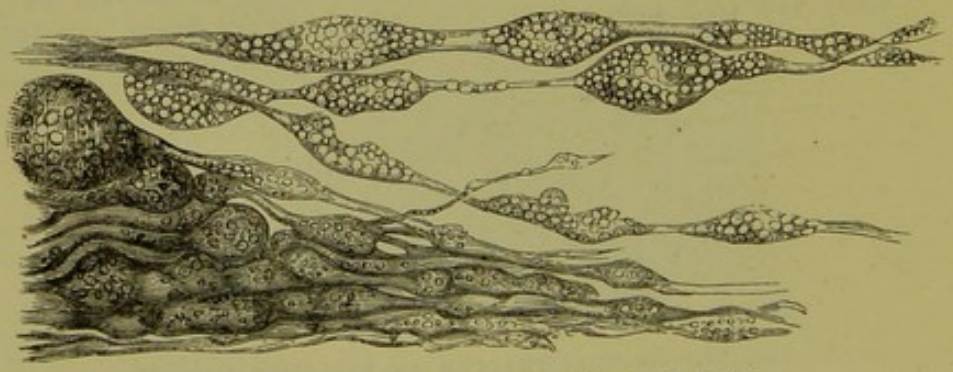
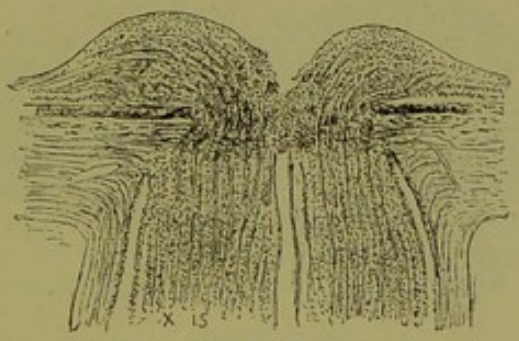


Fig. 15.—Degeneration of Nerve Fibres.

From the substance of an inflamed papilla in a case of tumour of the lower part of right middle cerebral lobe. Highly magnified. (After Pagenstecher and Genth.)

(Figs. 9 and 17). Similar corpuscles lie in greatly increased numbers between the bundles of nerve fibres. Some of these are nuclei belonging to a system of connective-tissue fibres which run at right angles to the nerve fibres (indicated in Figs. 8 and 11). These fibres may be themselves swollen. (4) The nerve fibres present changes, which contribute, in varying degree, to the production of the swelling. They are irregularly thickened, and the enlargements may be varicose, moniliform, or knob-like (Fig. 14), often containing granules or fatty globules from degeneration of the myelin. The swellings may attain a large size, as in the accompanying figure (Fig. 15). Free aggregations of fatty globules and granules may also be found, commonly enclosed in a cell wall ("granule-corpuscles") (Fig. 13); they may assume a colloidal appearance ("corpora amylacea"). Many of these are simply



Optic Neuritis in a Case of Cerebral Tumour.

Fig. 16.—Section through the centre of the papilla, showing the swelling of the outer part and a central depression, almost to the choroidal level. The nerve fibres can still be traced, separated by leucocytes. The same infiltration is to be seen in the nerve. The sheath is not distended, but its lining membrane is infiltrated with leucocytes.

Fig. 17—The same papilla near the edge. On the left the deeper layers of the retina are seen thrown into folds. (See p. 64.)

the detached degeneration-swellings of the nerve fibres. They are best seen in glycerine preparations. These products of degeneration give rise, by their aggregation, to the larger white spots seen with the ophthalmoscope. (See Fig. 7.) Other spots are apparently due to aggregations of leucocytes.

The vessels may have their walls thickened by nucleated tissue, and sometimes by a clear, finely fibrillated substance (Fig. 22).

The vessels do not usually present any evidence of compression in the sclerotic ring, but commonly appear to be narrowed, often considerably, in the thickest part of the swelling, and the veins are again enlarged as they pass down the sides. The veins are usually very large, the arteries narrow. The former, after curving down the sides of

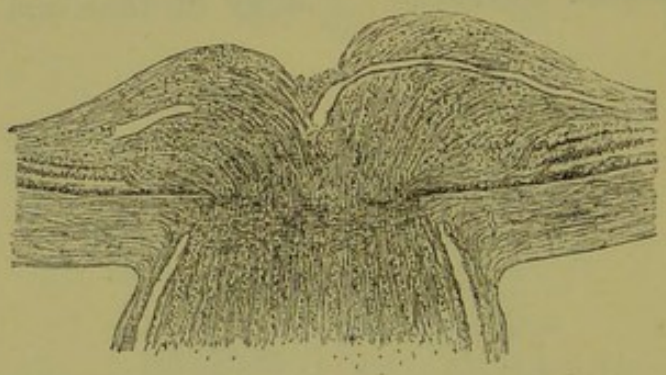


FIG. 18.—SECTION OF THE PAPILLA IN A CASE OF CEREBRAL TUMOUR.

There is considerable swelling, greater on one side. The commencement of the retina is displaced some distance from the edge of the sclerotic ring. Infiltration of leucocytes in the papilla and nerve-sheath, but the latter not distended. (× 20.)

FIG. 19.—SECTION THROUGH THE MIDDLE OF THE SAME DISC.

The central depression remains, although much narrowed. The central vein is seen divided longitudinally. Neither in the sclerotic ring nor behind it does the vein present any trace of compression. (× 8.)

the swelling, descend into the substance of the swollen retina, even into the nuclear layers, and rise again into the layer of the nerve fibres. Sometimes two such curves may exist (Fig. 23). The retina is displaced from the edge of the choroid, often as far as a millimetre from the sclerotic ring. Its layers at the commencement usually present considerable change. The nerve-fibre layer is thickened by a slighter degree of the changes which cause the swelling of the disc. The nuclear layers are increased in thickness and often blended together, and the nuclei more or less separated and grouped

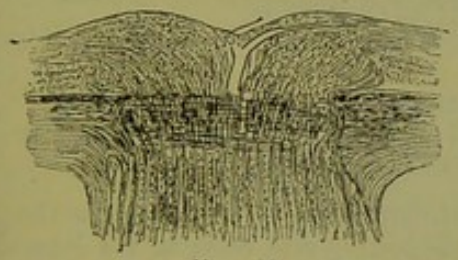


Fig. 20.

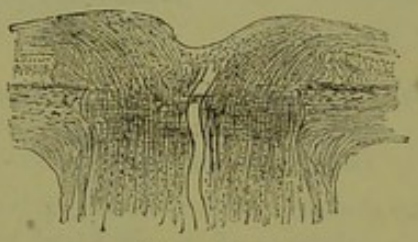


Fig. 21.

Figs. 20 and 21.—Sections through the Papilla in a Case of Optic Neuritis due to Chronic Cerebritis.

(Case published by Dr. H. Jackson in "Ophth. Hosp. Rep.," vol. viii. p. 445.) The papilla is slightly swollen, and has displaced the retinal layers. In Fig. 20 a vein is seen becoming compressed in passing through the inflamed retina, but it will be noted that in Fig. 21 there is no sign of compression, as the central nerve passes through the sclerotic ring. (× 15.) See also chapter on "Softening of the Brain."

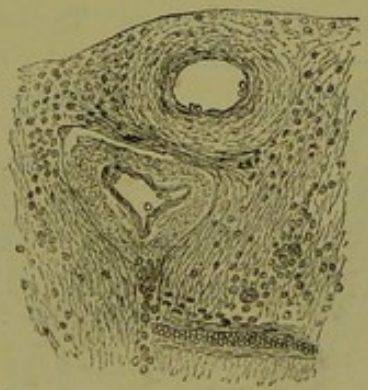


Fig. 22.—Part of a Section of an Inflamed Papilla in a Case of Optic Neuritis.

An artery (below) and a vein (above) exhibit thickening and fibrillation of their outer coats. Below is a small vessel showing similar changes. The surrounding tissue is infiltrated with leucocytes. (× 100.)

into vertical columns by the displaced fibres of Müller (Fig. 9). The retina may present (as here) slight curves due to its displacement, most marked in its outer (deepest) layers, and effecting detachment of the retina, the space between the bacillary layer and choroid being occupied by serum. These curves may be visible with the ophthalmoscope as pale bands, parallel to the edge of the papilla (Pl. VII. 1).

Fig. 23.—Section through the Swollen Papilla in a Case of Old Chronic Meningitis, with Inflammatory Growths in the Convexity of the Brain. (See Pl. VI. 2 and Fig. 33.)

At the edge of the swelling a large vein forms two vertical curves in the substance of the thickened retina, the lower curve reaching the inner nuclear layer. The retinal layers are displaced. On the right side the pigment-epithelium has disappeared in the portion from which the retina has been pushed away; on the left side the epithelium persists in this situation. (× 15.)

Fig. 24.—Section through the Retina,

Some distance from the disc in the same case. The vein occupies two-thirds of the thickness of the retina, and in one or two places has encroached on the nuclear layers. (× 50.)

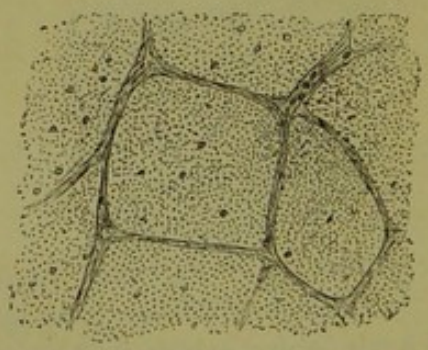


Fig. 25.—Section through a healthy Optic Nerve.

For comparison with the subsequent figures.

The pigment-epithelium may persist up to the edge of the sclerotic, or it may disappear in the area from which the retina has been displaced (Fig. 23). Often the choroid undergoes atrophy close to the edge of the sclerotic.

The changes in the papilla always become much slighter at the sclerotic ring, and may appear to cease there. Commonly, however, large numbers of nuclei lie among the nerve bundles in and just behind the lamina cribrosa, where such nuclei are in health most abundant. The sclerotic ring may appear distended, the nerve tissue occupying closely its funnel-shaped area. The appearance of distension is partly, if not entirely,

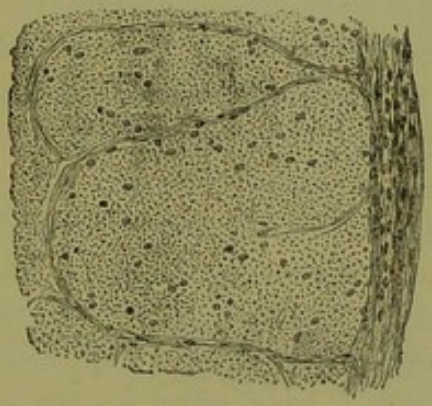


FIG. 26.—TRANSVERSE SECTION THROUGH THE OPTIC NERVE HALF AN INCH BEHIND THE EYE.

In a case of early optic neuritis. Thickening and infiltration of sheath. Very little change at present in the nerve. (× 150.)

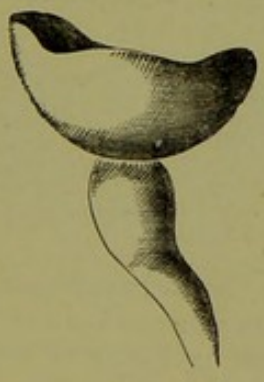


FIG. 27.

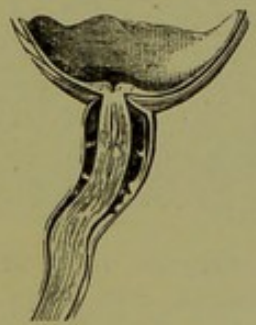


FIG. 28.

POSTERIOR SEGMENT OF EYEBALL AND OPTIC NERVE.

From a case of chronic traumatic meningitis, showing the distension of the sheath of the nerve and the swelling of the papilla. (Natural size, after Pagenstecher and Genth.)

due to the shape of the ring, as may be seen by comparing Fig. 17 with Fig. 23. In the latter the appearance of excavation of the edge of the sclerotic is present on the right side only, and an interval exists between it and the nerve fibres, occupied only by the fibres of the lamina cribrosa.

The sheath of the optic nerve is often distended with fluid, sometimes slightly, sometimes considerably. The distension is greatest a short distance behind the eye, and narrows close to the sclerotic, having thus a pyriform shape (Fig. 27). In cases of old neuritis the sheath may be enlarged but empty, showing previous distension. Microscopically the nerve may appear normal, the nuclear increase near the lamina cribrosa being absent farther back. More commonly signs of inflammation may be traced throughout the nerve; the nuclei are increased in quantity, its trabeculæ thickened and the vessels distended (Figs. 29 and 30). The inner sheath is often crammed with nuclei, and the connective tissue between the inner and outer sheath increased (Fig. 26). The nerve fibres may present evidence of degeneration (Figs. 29 and 30). These changes, slight or considerable, may often be traced back as far as the chiasma, in front of which they are sometimes much

FIG. 29.—SECTION THROUGH THE OPTIC NERVE, JUST BEHIND THE SCLEROTIC.

(Pl. III. 3). The nerve fibres present only an irregular granular appearance, the axis cylinders being no longer demonstrable. The sheath presents many compressed nuclei. (× 120.)

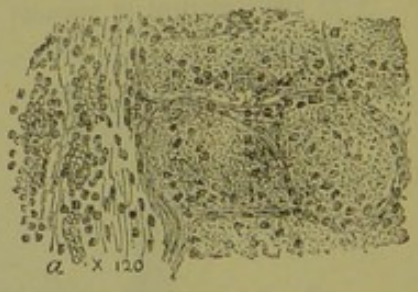


FIG. 30.—TRANSVERSE SECTION THROUGH THE SAME OPTIC NERVE, JUST IN FRONT OF THE COMMISSURE.

The sheath of the nerve (on the left) contains dilated vessels, and large numbers of leucocytes, which are also very abundant in the interfascicular septa. (× 120.)

more intense than anteriorly, and are most intense near the surface of the nerve. This is seen especially in cases of meningitis, and affords evidence of extension of inflammation from the meninges. An increase of nuclei is sometimes to be traced into the chiasma, and even into the optic tract (Fig. 32), where the corpuscles may even be aggregated into groups that have been termed "miliary abscesses" (Fig. 33).

During the progressive subsidence to atrophy, there is a diminution of the cellular elements in the papilla, probably, in part, in consequence of their transformation into fibres. The products of the degeneration of the nerve fibres are slowly removed. Ultimately the substance of the papilla appears to consist of a felty mass of interlacing fibres sprinkled with nuclei, in which at last scarcely any indication of nerve fibres is to be traced.

Symptoms.—Subjective symptoms may be entirely absent,

Fig. 31.—Longitudinal Section of the Optic Nerve, from a Case of Optic Neuritis,

Showing the irregular outline of the degenerating nerve fibres, and the infiltration of leucocytes between the fasciculi. (× 120.)

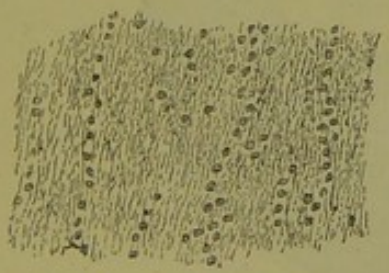


Fig. 32.—Longitudinal Section through the Optic Tract, in a Case of Optic Neuritis.

There is increase in the connective tissue corpuscles between the fibres (× 150.)

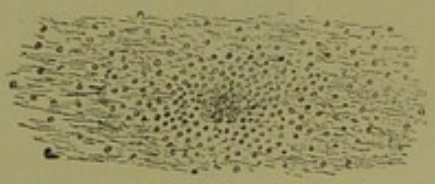


Fig. 33.—Section through the Optic Tract in a Case of Chronic Meningitis.

Shows aggregation of leucocytes into a "miliary abscess," such as is seen in the medulla in cases of hydrophobia (× 100.)

even when the inflammation of the papilla is of considerable intensity, as was first pointed out by Hughlings-Jackson. Vision may be unimpaired—acuity and colour-vision being perfect, and the field unrestricted. An increase in the size of the blind spot may usually be ascertained by mapping it out with the perimeter, but of this the patient is unconscious. The degree of neuritis which may exist, with no impairment of acuity of vision, is remarkable. In the cases shown in Pl. I. 4, 6, III. 5, IV. 1, 3, V. 1, 2, VI. 4 and 5, when the drawing was made, the acuity of vision was scarcely or not at all impaired. It is often said that "descending neuritis" causes much earlier affection of sight than limited intra-ocular papillitis. But acuity of vision may be unimpaired even with a considerable degree of descending neuritis. In more intense cases, however, sight is impaired or lost, and this constitutes the chief subjective symptom. Photophobia and pain in the eye are very rare in optic neuritis. Pain in the head may occur in cases of apparently primary papillitis: it is, of course, a very common accompaniment of symptomatic inflammation, but

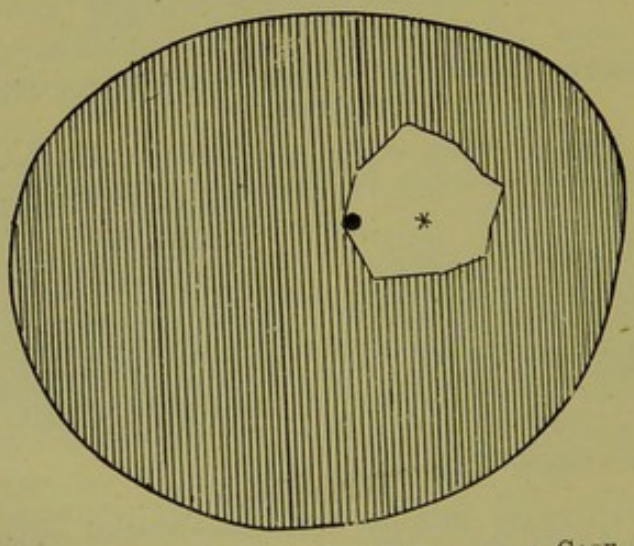


FIG. 34.—DIAGRAM OF THE FIELD OF VISION IN A CASE OF SUBSIDING OPTIC NEURITIS IN CEREBELLAR TUMOUR, LEFT EYE.

The outer boundary of the figure is the limit of the average normal field. Vision was lost in the shaded area, preserved only within the inner line around the fixing point, the position of which is indicated by the asterisk.

is then generally to be accounted for by the intra-cranial disease.

The affection of vision usually occurs earlier in the one eye than it does in the other. It may come on rapidly or slowly; never suddenly. Sometimes the rapidity of its progress may be great; sight may fail completely in the course of a few days.

Restriction of the visual field usually accompanies considerable change in the acuity of vision. It may be extensive, and often reaches its height during the stage of subsidence. Only a small area around the fixing point may remain, as in the diagram (Fig. 34), from a case of subsiding neuritis in cerebellar tumour. Occasionally the limitation of the field of vision may be irregular, one part being more or less affected than the rest, as in Fig. 35, in which the upper part only is restricted. In some cases a change in the field of vision, due to the intra-cranial disease, may accompany the peripheral limitation due to the optic neuritis, as in the diagrams (Figs. 36 and 37) of the fields of vision in a case in which hemianopia, owing to the intra-cranial disease, accompanied the peripheral limitation.

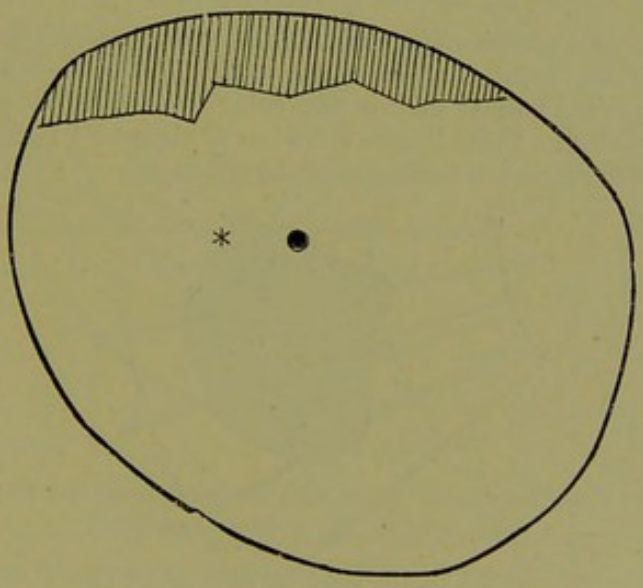


FIG. 35.—DIAGRAM OF FIELD OF VISION, SHOWING LIMITATION ABOVE ONLY.

From a case of unilateral optic neuritis, probably due to cerebral syphiloma. There was amblyopia, but no neuritis of the other eye. Both discs subsequently became atrophied.

Not unfrequently there is marked peripheral amblyopia, and a small object cannot be recognized in the periphery, although a large object, as the hand, is well seen. The increase in size of the blind spot is proportioned to the size of the papillary swelling. The accompanying diagram (Fig. 38) shows its area in a case of optic neuritis figured in Pl. IV. 3. It is a little, but not much, larger than normal.

When there is distinct amblyopia there may be a defect in

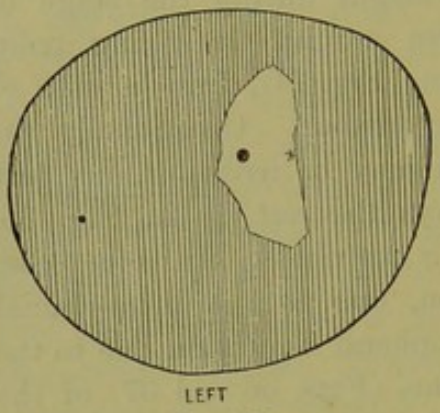


Fig. 36.

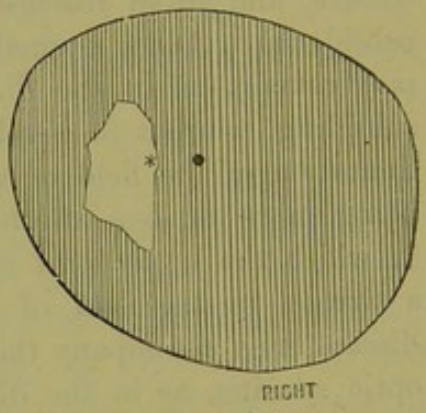


Fig. 37.

Diagrams of the Fields of Vision in a Case of Hemianopia and Double Optic Neuritis.

Probably due to a cerebral syphiloma. The asterisk represents the fixing point, the dot the position of the blind spot. The outer boundary of the shading is the normal limit of the field, the shading the area in which sight was lost. There is seen to be loss of the whole right half of each field, with concentric limitation of the left halves.

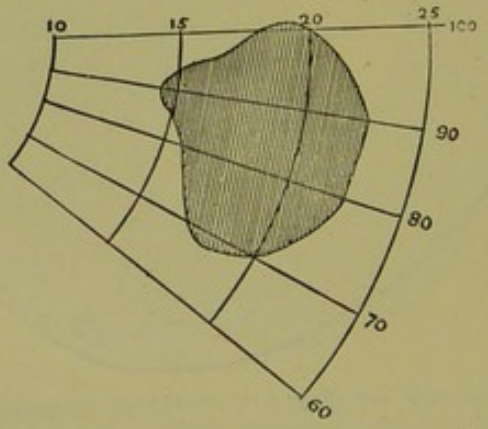


Fig. 38.—Diagram of the Blind Spot (Shaded Area) in a Case of Optic Neuritis.

From a case of tumour (probably a syphiloma) in the left ascending parietal convolution (see also Pl. IV. 3).

colour-vision, and the latter may exist even when acuity of vision is very little impaired. The order of loss is sometimes (as in atrophy, *q. v.*) that of the normal peripheral arrangement of the colour fields in the accompanying figure (Fig. 39), red and green being lost before yellow and blue. Thus in a case under the late Dr. Radcliffe, of a girl aged eleven, who had optic neuritis of both eyes, there was little limitation of the field for white. The only colour which she could name accurately was yellow. Light shades of other colours were called white, deep shades black. More frequently, however, the loss is irregular. In three cases I have seen yellow alone lost. In a case of severe neuro-retinitis due to chlorosis, at one time, yellow was alone lost in one eye, and in the other eye yellow, blue, and green were lost, red being seen; and recovery was in the order of the fields, the yellow last. Now and then colour-vision may be little affected, even when there is considerable peripheral limitation of the field of vision.

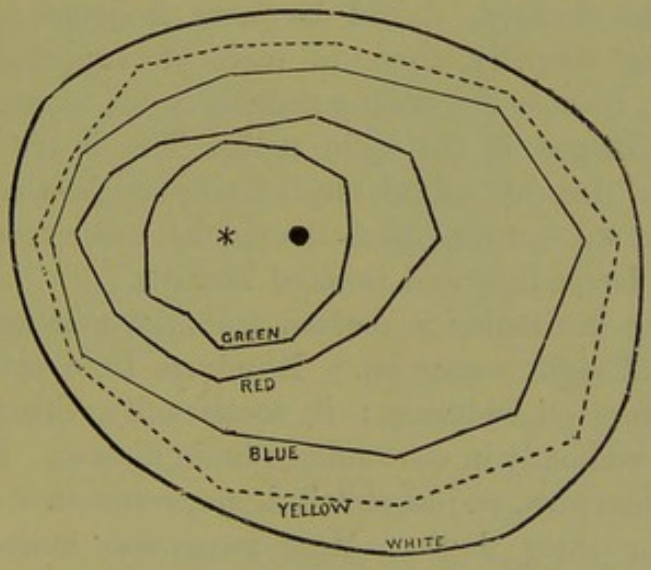


FIG. 39.—DIAGRAM SHOWING THE FIELDS OF COLOUR-VISION IN A NORMAL EMMETROPIC EYE ON A DULL DAY.[1]

The fields are each rather smaller than on a bright day. The asterisk indicates the fixing point, the black dot the position of the blind spot. (Usually the blue field is larger than the yellow.) See the section on "Atrophy of the Optic Nerve."

[1] I am indebted to Mr. Nettleship for the charts from which this diagram was made.

When sight is completely lost, the sensibility of the retina to electrical stimulation may or may not be impaired. It may be lost during blindness, and return with some recovery of sight.

It is very important to be aware as far as possible of the mechanism by which sight is impaired, since the prognosis must, in the main, depend thereon. The loss of sight which occurs in cases of idiopathic isolated papillitis is, of course, due to the process which can be seen with the ophthalmoscope. But the papillitis which occurs in intra-cranial disease may be accompanied with loss of sight due, not to the intra-ocular changes, but to mischief in the course of the optic fibres or in the centres with which they are connected. The first point to ascertain, therefore, is whether the amblyopia is due to the intra-ocular changes or to mischief farther back. It is not always possible to determine this point, but very often an accurate opinion may be formed.

Concomitant affection of sight from intra-cranial processes, it may be thought, should be more frequent in cases of descending neuritis, than in cases of supposed isolated papillitis; because descending neuritis is due to, and involves, organic changes in the optic path. This is true, but this distinction does not afford much assistance, because it is not often that we can rely upon the ophthalmoscopic distinction between descending and isolated neuritis.

Another distinction is derived from the manner in which the loss of sight comes on. Blindness from optic neuritis never comes on suddenly; it occasionally, though rarely, comes on suddenly in concomitant brain disease. From optic neuritis, however, as just stated, it may come on in the course of two or three days. More important indications are derived from the form in which sight is lost. A symmetrical hemiopic defect in the field (such as in Figs. 36 and 37) means an intra-cranial cause; and unsymmetrical lateral defect, especially a loss of the temporal halves (as Figs. 40 and 41), ordinarily means pressure on the chiasma, a very common cause of blindness in these cases, the pressure being exerted by a distended third ventricle. Complete loss of

sight of one eye, and loss of the adjacent half of the other field (as in Figs. 42 and 43), is hypothetically of cerebral origin.[1] A peripheral restriction of the fields usually means

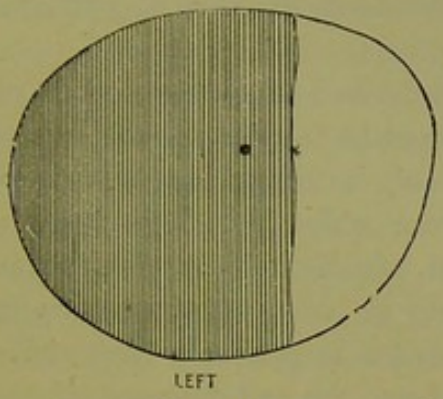


Fig. 40.

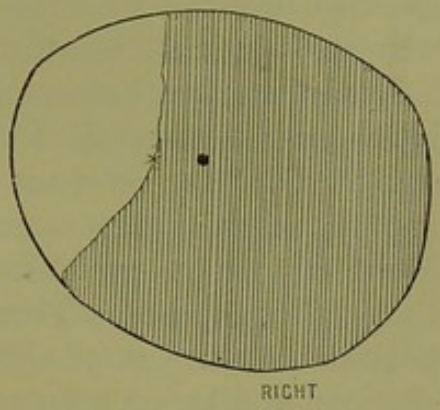


Fig. 41.

Diagrams of the Fields of Vision in a Case in which Sight was probably lost from Pressure on the Chiasma.

The shaded area indicates the part in which vision was lost—viz., the temporal portions of both fields,—the nasal portions alone persisting ("temporal hemianopia").

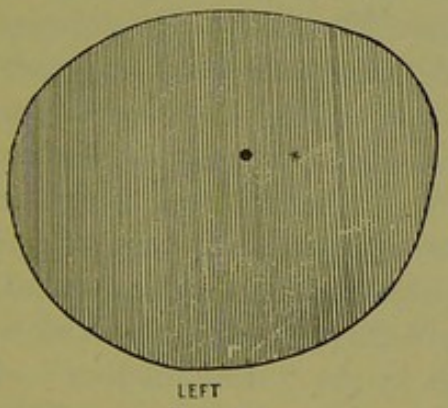


Fig. 42.

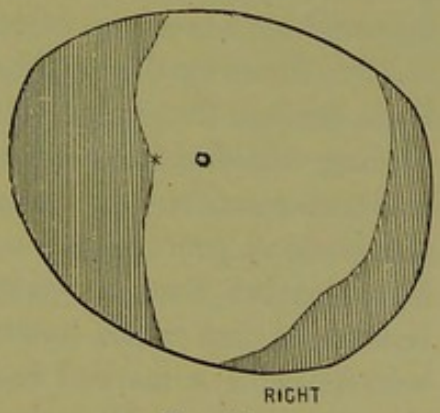


Fig. 43.

Diagrams of the Fields of Vision in a Case of Loss of Sight and Disease of the Right Cerebral Hemisphere.

Loss of the whole of the left field and of the left half of the right, with a little peripheral defect on the temporal (right) side.

[1] The loss may be explained on the unproved hypothesis of Charcot that there is a secondary decussation at the corpora quadrigemina, complementary to the approximate semi-decussation which certainly takes place at the chiasma. Thus an extensive lesion at the posterior part of one optic thalamus would destroy the fibres which had crossed to that side at both decussations (*i.e.*, all from the opposite eye) and those which were about to decussate at the corpora quadrigemina from the eye on the same side. The only uninjured fibres would be those from the inner half of the retina, on the

damage in front of the optic commissure, and, in most cases of intra-ocular neuritis, damage from the visible changes. A central scotoma is observed only when there is a conspicuous lesion at the macula lutea, or in cases of primary retro-ocular (axial) neuritis.

Lastly, important assistance is derived from the degree of intra-ocular damage—is it sufficient to account for the loss of sight? The question is one difficult in many cases to answer, and an approximate answer can only be afforded by a knowledge of the conditions on which the loss of sight, in these cases, depends. The study of intra-ocular neuritis in relation to affection of sight makes it probable that vision may suffer in two ways, apart from the involvement of the retina. The first is damage to the nerve fibres by the process of inflammation around them. We know that acute inflammation has a tendency to stop the conducting power of nerve fibres, apparently by some direct damage to their finer structure, and that the subsidence of the inflammation may be followed by a recovery of function. The second is by pressure on the fibres by the products of inflammation. This occurs both during the inflammation and while it is subsiding. During subsidence, the newly-formed elements are undergoing a transformation into contracting fibrous tissue. A similar compression probably also occurs during the height of the inflammation, from the excessive amount of tissue produced, because at that period loss of sight may often be observed to coincide with a marked increase in the "strangulation" of the swollen disc. Damage to vision from compression of the fibres during subsidence of the neuritis is very common and very serious. It may cause considerable damage to sight which has been unimpaired by the active neuritis, and it constantly succeeds and intensifies impairment by inflammatory compression during the active stage. The amblyopia which occurs during the height of the inflammation may lessen as the inflammation subsides, and be again renewed by the contraction of the new tissue as the subsidence of the swelling

same side as the cerebral lesion, fibres which had crossed at the chiasma to the opposite hemisphere.

advances. The latter constitutes by far the greatest danger, because the contraction which causes it continues, and increases for a long time, and the amblyopia due to it usually continues and increases until, and even after, the disc has reached its normal level. The progress of the amblyopia from this cause may, however, be interrupted by the recovery of fibres damaged only during the active inflammatory stage, and when the amount of new tissue formed is small in proportion to the irritative changes in the disc (as in many cases of syphilitic disease duly treated), a considerable inflammatory amblyopia may clear away and be followed by very slight consecutive defect. The latter is usually more or less permanent, but, after it has reached its height, considerable subsequent improvement does, in some cases, slowly occur (see p. 130).

Not unfrequently after a neuritis has subsided, with or without impairment of vision, a further loss of sight, complete or incomplete, may occur from intra-cranial causes without any fresh ophthalmoscopic appearances. It will thus be seen that a considerable disparity between the affection of sight and the course of the papillitis indicates the influence of retro-ocular mischief. A good illustration of this is afforded by the case figured in Pl. VII. 1 and 2, in which the papillitis was unilateral, the other eye presenting normal characters throughout; but the sight of both eyes failed after the subsidence of the papillitis (see Fig. 35).

Causes.—The most common causes of optic neuritis are encephalic diseases, and of these tumour is incomparably the most frequent. Neither the nature, size, nor the seat[1] of the tumour appears to exercise much influence on the occurrence of neuritis. The next most frequent cause is certainly

[1] From an analysis of cases made by Edmunds and Lawford it would appear that, *ceteris paribus*, tumours near the convexity of the brain are somewhat less liable to cause optic neuritis than those situated near the base. Cerebellar tumours seemed particularly prone to excite optic neuritis, often of a severe type. Of twelve cases of primary tumour of the cortical motor area, on the other hand, not one was associated with optic neuritis ("Trans. Ophth. Soc.," vol. iv. 1884, p. 172).

meningitis, and then come abscess of the brain, hydatid disease of the brain, and softening of the brain from vascular obstruction. In some cases it appears to result from an irritative process in the brain, revealed only by the microscope (see Part II., "Inflammation of the Brain"). It also accompanies, in rare cases, acute diseases of the spinal cord. Other causes, outside the nervous system, are—albuminuria, lead and tobacco poisoning, certain febrile diseases, anæmia (especially from loss of blood), and certain other morbid blood states. It may probably occur as an idiopathic affection, without obvious exciting cause, or from disturbances of menstruation, or exposure to cold. In all these cases it is, as a rule, double; now and then, in cerebral disease, and after acute febrile diseases and loss of blood, it may be single. Unilateral optic neuritis may result from mischief in the posterior portion of the orbit—inflammation or growth invading the optic nerve.

In the general diseases, such as albuminuria, lead poisoning, anæmia, &c., optic neuritis is often associated with encephalic symptoms. In a case of lead poisoning (Pl. VII. 6), the neuritis was associated with great mental excitement, and so also in a case of albuminuria (Pl. IX. 2), while in the similar case figured in Pl. IX. 3, intense headache was present. It seems probable that, in these cases, either the cerebral disturbance is concerned in the production of neuritis, or the neuritis and cerebral disturbance may be the indication of a general effect of the toxæmia on the nerve tissues.

Duration.—The duration of optic neuritis varies very much in different cases. The cases of most rapid course may reach their height in two or three weeks, maintain it for about the same time, and then subside. These are of two classes—the most trifling, and the most severe. The former are, for the most part, those which depend on a cerebral condition which soon subsides, such as a transient attack of meningitis, or syphilitic or scrofulous disease, which is influenced by treatment before the neuritis reaches its height.

Now and then, however, a neuritis rapidly subsides, although the cerebral disease progresses. But this is the rare exception. In these transient cases the subsidence may occupy the same time as the development—two or three weeks—and be complete; so that at the end of six or eight weeks the discs are again normal. In some very intense cases, such as that of apparently idiopathic neuritis figured in Pl. VIII., the development of the affection may be equally rapid, an intense degree of swelling being soon attained, and subsidence commencing in a few weeks. In these cases, however, the retrocession of the neuritis is always slow, and commonly occupies many weeks; often months pass before the edges of the disc are again perceptible. In two cases of cerebral abscess, where the pus was evacuated by an operation, the neuritis, which was extreme, subsided in a little more than a fortnight.

On the other hand, the course of neuritis may be so chronic that months, even a year, may pass without the least change in the condition of the discs being perceptible (for instances of this see cases 23, 24, and 26 in former editions of this book). Most cases of this extreme chronicity that have come under my observation have accompanied symptoms of brain disease which were not, in themselves, suggestive of "coarse" brain disease, tumour, &c. But in some cases an equally chronic neuritis may accompany tumour. That shown, for instance, in Pl. V. 1 and 2 presented no alteration, tested by comparison with the drawing, for eighteen months, and two years later was still marked, the red, congested half being similar in area, although on the clearer half the disc had become grey, and sight was lost. It is to be noted that in this case the symptoms of tumour, although intense, also progressed very slowly, and the chronicity of the neuritis probably may be taken as an indication of chronicity of the cerebral disease. The converse proposition, however, that all forms of very chronic brain disease entail a chronic form of neuritis, does not, by any means, hold good.

In most cases the duration of neuritis is intermediate between the extremes mentioned, reaching its height in

a month or two, and often remaining for some weeks or months with little change, and then subsiding. A rapid strangulation usually precedes subsidence at no long period, the products of inflammation perhaps checking the inflammatory process.

The Relation of Optic Neuritis to Encephalic Disease.—The first definite theory of the mechanism by which intra-cranial disease acts was put forward by von Graefe in 1859,[1] and further developed by him in 1866.[2] It was founded on the observation that in some cases of intra-ocular neuritis, with hæmorrhages, in cerebral tumour, no signs of inflammation were perceptible on naked-eye examination in the trunk of the optic nerve; whereas, in a case of meningitis in which the ophthalmoscopic changes had been less intense, inflammation of the nerve trunk was found by Virchow—inflammation which was naturally assumed to have been communicated to the optic nerve from the inflamed meninges, and to have descended the nerve to the eye. This condition von Graefe designated "descending neuritis," and gave as its characteristics a slight degree of change in the discs and a tendency to invade the adjacent retina. On the other hand, the cases of tumour, with great intra-ocular change, hæmorrhages, &c., and no evidence of inflammation in the optic-nerve trunks, he explained by the theory that they were due to the effect on the circulation of the eye of the increased intra-cranial pressure, which he assumed to be invariable in these cases, and to obstruct the return of blood from the eye by compressing the cavernous sinus. He suggested further that this mechanical effect was greatly intensified by the unyielding character of the sclerotic ring, which would act, he assumed, as a multiplier of the mechanical obstruction. In accordance with this view he applied to the condition of disc met with in these cases—considerable

[1] In a communication to the Société de Biologie of Paris in November, 1859 ("Gazette Hebdom.," 1859), and more fully described in the "Arch. f. Ophth.," vii. 1860, pt. 2, p. 58.

[2] "Arch. f. Ophth.," xii. p. 100.

swelling with hæmorrhage and vascular distension — the term "stauungs-papille" (*stauung*, a damming back), in distinction from the "descending neuritis." In this country, by the suggestion of Clifford Allbutt, the term "choked disc" has come into use as a synonym for "stauungs-papille."

It was soon pointed out as strange that an actual inflammation should result from a mechanical congestion, and as still more strange that the inflammation thus excited should remain limited so nearly to the papilla. But graver difficulties awaited this theory of the "stauungs-papille." It was found that the ophthalmoscopic signs of the two forms of neuritis could not always be relied upon. The condition supposed to be characteristic of descending neuritis was discovered, in some cases, to be but the first stage of that supposed to indicate mechanical obstruction. It was found, also, that when the character of one of the two forms was clear the necropsy might show the case to be really one of the other variety. Cases of this character were frankly published by von Graefe, although he still held that the distinctions were, in the majority of cases, accurate, and the supposed mechanism of the "stauungs-papille" effective. This theory, however, was destroyed in its substantive form in 1869 by the demonstration by Sesemann[1] that the communication between the supra-orbital and the facial veins was so free that the effect of pressure on the cavernous sinus was at once relieved, and did not cause more than a very transient fulness of the retinal veins, and that even obliteration of the cavernous sinus produced no intra-ocular changes. This has since been well corroborated, as, for instance, by a case recorded by Hutchinson, in which no distension of the retinal veins was produced, although the cavernous sinus was completely obliterated by the pressure of an aneurism. It has, indeed, been said that the openings from the orbital into the facial vein are often larger than the communication with the cavernous sinus.

[1] "Reichert u. Du Bois Reymond's Archiv," 1869, p. 154.

It was discovered by Schwalbe[1] that the subvaginal space around the optic nerve is, at the optic foramen, continuous with, and can be injected from, the subdural space around the brain.[2] This gave significance to some earlier observations of Stellwag von Carion[3] and Manz[4] that the sheath of the nerve may be distended in optic neuritis from tumour and meningitis. The two facts suggested to Schmidt[5] that intra-cranial pressure may influence the intra-ocular termination of the optic nerve by this mechanism, since, as already described, the distension of the sheath is greatest just behind the globe. The theory received support from Manz,[6] who showed how frequent distension of the sheath is in optic neuritis, and believed it to be invariable in cases of increase of intra-cranial pressure or increase of subarachnoid fluid. He urged that the simple pressure on the nerve and vessels might cause the intra-ocular changes, and endeavoured, by experiment on animals, to demonstrate this effect of the vaginal distension. Injections into the subdural space passed into, and distended, the sheath, and caused fulness of the retinal veins, and in some cases transient redness and swelling of the papilla.

Schmidt, however, found that a coloured liquid injected into the sheath passed into lymph spaces in the nerve at the lamina cribrosa, and he suggested that neuritis is produced, not by the simple pressure outside the nerve, but by the influence, perhaps irritation, of the liquid passing into these lymph spaces. The theories of Schmidt and Manz have been largely accepted in Germany as affording the most satisfactory explanation of the origin of optic neuritis. Leber,[7] while adopting the view that the distension of the

[1] „ Centralblatt f. Med. Wiss.," 1869, p. 465. "Arch. f. Mikroskop. Anat.," Bd. vi. 1870, p. 1.

[2] It has been stated by Parinaud that the communication is with the subarachnoid, not with the subdural space ("Ann. d'Oculistique," vol. lxxxii. 1879, p. 25).

[3] "Ophthalmologie," vol. ii. 1856, p. 612.

[4] "Zehender's Monatsbl.," vol. iii. 1865, p. 281.

[5] Of Marburg, now Schmidt-Rimpler. "Arch. f. Ophth.," vol. xv. 1869, p. 193.

[6] "Deutsch. Arch. f. Klin. Med.," vol. ix. 1871, p. 339.

[7] Discussion at the International Medical Congress, London, 1881.

sheath is the immediate excitant of neuritis, doubts the theory of Manz, that the fluid acts by mechanical pressure, and rejects the effect on the lymphatic spaces assumed by Schmidt, on the ground that his own and other investigations have failed to confirm the asserted communication of these spaces with the sheath. Leber suggests that the fluid in the sheath excites neuritis, by conveying pathogenic material to the optic nerve behind the eye. Deutschmann[1] has recently published experimental evidence in favour of Leber's view, and in opposition to the theory of "choked disc" from the distension of the nerve-sheath.

It was suggested in 1863 by Hughlings-Jackson,[2] that intra-cranial tumour causes optic neuritis by its irritating effect, acting as a "foreign body," and this view was supported a little later by Brown-Séquard, who compared the origin of neuritis in intra-cranial tumour to the production of atrophy of the optic nerve by a distant source of irritation. It was more precisely formulated by Benedikt[3] in 1868, by ascribing the mechanism to the vaso-motor nerves, and it is sometimes termed his theory.[4] This view assumes that the tumour acts as a source of irritation, producing a reflex influence through the vaso-motor nerves upon the optic disc, and thus leading to its inflammation. It has been rejected by most writers on the grounds stated by Leber,[5]—that it involves a mechanism not known to exist, and a complex relation of the optic nerve to all parts of the brain difficult to conceive; and by Clifford Allbutt, on the ground that he has failed to find around tumours the signs of irritation. The theory is, however, still held by Hughlings-Jackson as that which best explains the phenomena of

[1] "On Optic Neuritis, especially the so-called Choked Disc, and its connection with Brain Diseases." Jena, 1887, and "Oph. Rev.," vol. vi. 1887, p. 107.

[2] "Ophth. Hosp. Rep.," vol. iv.

[3] "Allg. Wien. Med. Zeit.," 1868, No. 3.

[4] Schneller, in 1860, put forward a similar theory when he suggested that some retinal changes in intra-cranial disease might be due to a "primary affection of the centres of those nerves which regulate the course of the blood in the ocular vessels."—"Arch. f. Ophth.," Bd. vii. 1860, I. p. 71.

[5] In "Graefe u. Saemisch's Handbuch," Bd. v.

neuritis; he has always urged that the occurrence of optic neuritis is not related to increased intra-cranial pressure. Galezowski believes that neuritis is always descending, and first maintained, contrary to previous observers, that the intra-ocular change is in all cases the visible manifestation of an inflammation propagated by continuity from the brain. Edmunds and Lawford are of opinion that optic neuritis, when due to an intra-cranial cause, is secondary to basal meningitis, and that the inflammation reaches the substance of the nerve-trunk through its sheath.[1] Lastly, Parinaud[2] asserts that neuritis is invariably the effect of distension of the ventricles of the brain, which causes general cerebral œdema, and of this both the distension of the sheath and the papillitis are equally part.

The clinical and pathological evidence bearing on these views may be briefly reviewed.

The first point to be borne in mind is that optic neuritis limited to, or at least most intense in, the optic papilla, may occur without any obvious intra-cranial disease. The intense neuritis shown in Pl. VIII. 1 was apparently a primary papillitis, involving the retina only secondarily, due to anæmia. This patient had no symptom of cerebral disease, save some headache, during two years she remained under observation. The neuritis reached its height in about a fortnight from its commencement, a rapidity which is seen in neuritis from intra-cranial disease only in the most acute cerebral affections; this circumstance, with the absence of cerebral symptoms, excludes the supposition that there existed intra-cranial disease. Limited papillitis is now known to occur in simple anæmia. From these considerations it seems to follow that the intra-ocular termination of the optic nerve is a structure, for some reason which we do not know, peculiarly prone to suffer inflammation. The common localization of the inflammation to the papilla points also to the same fact.

[1] "Trans. Ophth. Soc.," vol. i. p. 111; vol. iii. p. 138; vol. iv. p. 172; vol. v. p. 184; vol. vii. p. 208.

[2] See "Graefe u. Saemisch's Handbuch," Bd. v.; "Ann. d'Ocul.," t. lxxxii. p. 5.

The facts of medical ophthalmoscopy certainly make it difficult to connect papillitis with increase of intra-cranial pressure. If we consider the cases in which intra-cranial pressure is raised to the highest point it ever reaches—chronic hydrocephalus—we find optic neuritis the rare exception, and, when it occurs, never intense. The difficulty cannot be met by attributing it to the slowness with which the pressure is raised, because the growth of many tumours, which cause intense optic neuritis, is equally slow.

On the other hand, as I have many times seen, in cases of tumour with neuritis there may be no sign of increased intra-cranial pressure during life or after death. "In these cases of vast tumours, the optic neuritis does not differ from that caused by small tumours at the vertex of the brain, which cannot exercise pressure of any consequence at the base." "The neuritis runs through its stages, and the swelling of the discs subsides, although the intra-cranial pressure goes on increasing."[1] There may also be signs of increased pressure in tumour without optic neuritis. But, while pressure upon the cavernous sinus cannot be regarded as the cause of neuritis, its influence on the retinal vessels cannot be altogether excluded. Experiments show that a quickly induced increase of pressure within the skull causes a transient distension of the retinal and papillary vessels. In tubercular meningitis (*q.v.*) Garlick's careful observations[2] have shown that, while papillitis is not, fulness of veins is related to an increased intra-cranial pressure.

In the course of a descending neuritis the distension of veins may be very great, as I have several times observed both during life and after death (see Figs. 6 and 24). In tumour the veins at first, and often throughout (when the neuritis does not reach a considerable degree of intensity), are little above the normal size, and present no tortuosities except those which are given them by the prominence of the papilla. The great distension of veins and narrowing of

[1] Hughlings-Jackson: Lecture on Optic Neuritis, "Med. Times and Gaz.," 1871, vol. ii. p. 581.

[2] "Med.-Chir. Trans.," vol. lxii. 1879, p. 441.

arteries occur when the inflammation has reached a certain degree of intensity. This points to the neuritic process in the papilla as causing the strangulation by pressure on the vessels. This view is entirely borne out by pathological investigation. I have never been able to discover any evidence of constriction of the vessels in the sclerotic ring or behind it. Their calibre here is always uniform (see Figs. 16, 19, 21, and 44). This statement is based on a very careful search for any evidence of such compression in a number of cases of papillitis from various intra-cranial diseases. In one case only was there an appearance of narrowing, and in this, from the unaltered course of an adjacent vessel, it was evidently due to a slight alteration in the position of the vessel at the spot, in consequence of which the sections ceased to pass through its widest part. It is always in front of the sclerotic, in the substance of the swollen papilla, that the vessels present conspicuous constriction — are pressed upon, and have their walls thickened by new tissue (Figs. 10, 20, 22, 23, &c.). Further, the most intense signs of "strangulation" may be seen in cases in which, as in that of neuro-retinitis due to chlorosis (Plate VIII. 1), there is reason to believe there is no intra-cranial disease; and in the case of chronic cerebritis, quoted at p. 89, in which there was no intra-cranial condition which could cause any mechanical effect, the intra-ocular signs of constriction and mechanical congestion were very marked.

Distension of the optic sheath is certainly very frequent in cases of optic neuritis. It is not, however, as has been alleged, invariable, either in cases of cerebral tumour with optic neuritis, or in conditions of increased intra-cranial pressure. It may be absent in tumour of the brain with characteristic neuritis; for instance, in one case a large glioma of the right frontal lobe, with hæmorrhage into it, was attended by optic neuritis, but with no distension of the sheath. This may also be absent in tumour with internal effusion; great distension of the lateral and third ventricles was caused in another case by a tumour near the corpora

quadrigemina,—there was optic neuritis but no distension of the sheaths. A case of tumour of the cerebellum with optic neuritis and no distension of the sheaths has been recorded by Nettleship.[1] In another case of old neuritis, due to a tumour occupying the whole of the third ventricle and inter-peduncular space, extending in front of the optic commissure and causing enormous distension of the lateral ventricles, the optic sheaths carefully examined in situ were quite empty. But they were loose, and had evidently been much distended. This case suggests that pressure at the base of the brain may even be incompatible with continued distension of the sheath. Distension was absent in a case of neuritis from cerebral abscess recorded by Carrier,[2] and in a case of double neuro-retinitis, apparently secondary to cerebral hæmorrhage, which has been recorded by Gemuseus.[3] In tubercular meningitis, again, the condition to which the distension of the sheath appears to be related is not distension of the ventricles, or increased intra-cranial pressure, but increase of the subarachnoid fluid, and it bears in this disease certainly no relation to the occurrence of neuritis. Of six cases with changes in the papilla, in which the state of the optic sheaths was carefully noted by Dr. Garlick, in four, in which excess of subarachnoid fluid was absent, the sheath was normal, although in several there was great distension of the ventricles, while in the remaining two cases, in which there was an excess of subarachnoid fluid, there was also dropsy of the optic sheath. A case of neuritis in tubercular meningitis, without distension of the sheath, has also been described by Edmunds.[4] It has been suggested that the fluid may be formed within the sheath itself, being prevented from passing backwards to the cranium by the intra-cranial pressure. If the sheath of the optic nerve is the chief lymph-channel by which fluid is conveyed away from the eye, its distension in optic neuritis, by

[1] "Path. Trans.," 1880, p. 252.
[2] "Philadelphia Med. Times," Jan. 29, 1880.
[3] "Klin. Monatsbl. f. Augenheilk.," 1880, p. 380.
[4] "Trans. Ophth. Soc.," vol. i. 1881, p. 112.

fluid escaping from the papilla, is intelligible. But this fact is, at least, uncertain (see above, p. 81). Moreover, the case of tumour in the third ventricle, referred to in the previous page, seems opposed to this theory. The optic sheaths had been greatly distended, but they had become empty, apparently in consequence of the pressure on the front of the base, by the large tumour in the anterior part of the third ventricle, cutting off the communication with the subarachnoid space. If the fluid were derived from the eye, this influence should have increased the distension of the sheaths, instead of causing them to become empty. There is thus strong reason to believe that the fluid in the sheath of the optic nerve passes into it from the subarachnoid space. The absence of obvious excess of the subarachnoid fluid in some cases, as in an instance mentioned by Edmunds,[1] is not of much weight as evidence against this conclusion, because a general increase of intra-cranial pressure (*e.g.*, by ventricular effusion) which would assist in forcing the fluid into the sheaths, would at the same time tend to remove it from the base of the brain where its amount is estimated.

Of the frequent association of dropsy of the sheath and optic neuritis there can be no doubt, but of the relation of one to the other, little evidence has yet been adduced. Manz admits that there are probably various kinds of effusion into the sheath, and that all may not lead to neuritis. Parinaud has asserted that it is common in cases of pulmonary obstruction, as in croup. It is certain, too, that distension of the optic nerve-sheath may occur even in meningitis without causing neuritis, as in a case recorded by Broadbent. But such cases prove little, because the duration of the dropsy may not have been sufficient for the inflammatory changes to arise. The occasional occurrence of papillitis without it—a fact which is well established—shows that it is not the invariable, and suggests that it is not the chief, mechanism by which papillitis is produced. But, it is probable that, although not the chief cause, it may still exercise an important influence on the process.

[1] "Trans. Ophth. Soc.," vol. i. 1881, p. 112.

In examining the trunk of the optic nerve behind the eye, in cases of papillitis from cerebral tumour, I have found the optic nerve to present traces of inflammatory change, increase of nuclei and connective tissue, much more frequently than

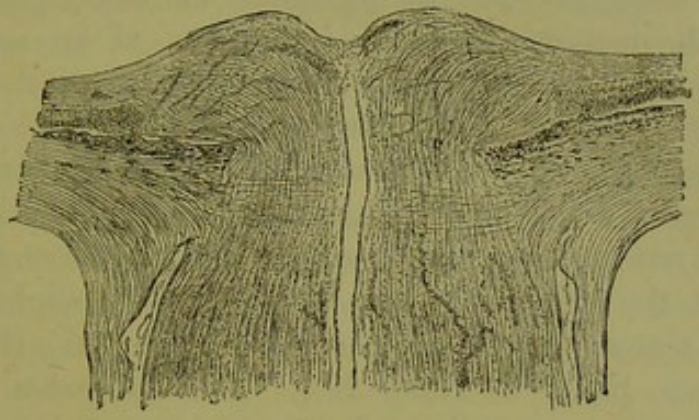


Fig. 44.

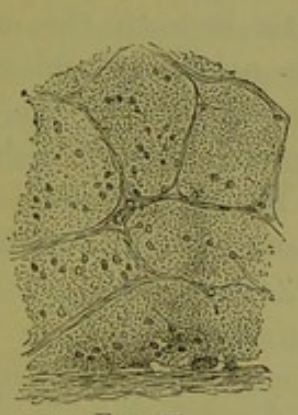


Fig. 45.

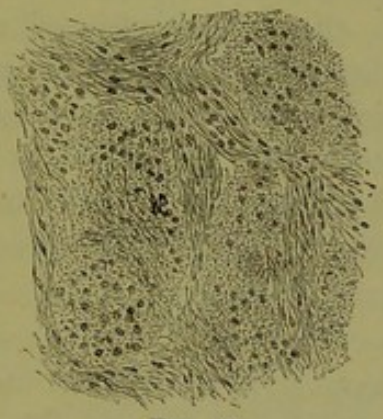


Fig. 46.

Sections from a Case of Optic Neuritis
Due to a tumour in the frontal lobes.

Fig. 44.—Section through the Disc. Swelling of the papilla, displacement of the retina outwards. Aggregation of leucocytes along the course of the vessels, thus indicating their position. No sign of compression of vein when passing through the sclerotic ring. No distension of the sheath of the nerve (× 15).

Fig. 45.—Part of a Transverse Section through the Optic Nerve, midway between the Globe and the Optic Foramen. Increase of leucocytes and some degeneration in the nerve fibres (× 100).

Fig. 46.—Transverse Section just in front of the Commissure. The bundles of nerve fibres are separated by much newly formed fibrous tissue, which is encroaching on the fasciculi of the nerve. The nerve fibres are degenerated, and many leucocytes are scattered among them (× 100).

has been stated by other observers. The changes were especially marked towards the periphery of the trunk and in the pial-sheath. In not one case examined could the nerves be said to be in a perfectly normal state. The significance of the changes is open to question. They may possibly be regarded, in some cases, as indications of an ascending neuritis. But in some they were most intense in the neighbourhood of the optic commissure (compare Figs. 45 and 46), and there was evidence that a neuritis had taken place there by extension from the meninges. In several cases in which this was most distinct, the change in the optic nerve, mid-way between the commissure and the eye, was so slight that it might almost have been passed as normal (Fig. 45. See also Figs. 26 and 29). The extension to the nerve was very distinct in two cases of intra-cranial tumour under my care. In one there was distinct, although very slight, evidence of meningitis beneath the orbital lobules, which had evidently, by the naked-eye and microscopic appearances, extended to the nerves. In the other case (Fig. 44) the papillitis was considerable, and such as is most common in cerebral tumour. The changes in the optic nerves in the middle of their course were slight but distinct

FIG. 47.

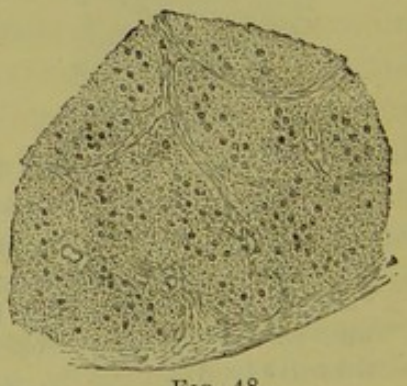


FIG. 48.

SECTIONS THROUGH THE OPTIC NERVE IN A CASE OF CHRONIC CEREBRITIS (× 100).

FIG. 47.—Just behind the globe, containing many leucocytes within the fasciculi.

FIG. 48.—Just in front of the commissure. There is more infiltration with leucocytes, and the connective tissue septa are more thickened and irregular.

(Fig. 45). In front of the optic commissure (Fig. 46) the changes from old inflammation were intense. No sign of adjacent meningitis was noted at the post-mortem examination, but there were old adhesions over the tumour on the upper part of the frontal lobe. In this case it seems probable that descending neuritis had taken place, and that the link between the intense neuritis behind, and the intense papillitis in front, was the slight change in the trunk of the nerve. Hence it seems that a very slight amount of descending change may lead, in cerebral tumour, to an intense papillitis. With this case may be compared another, of a man who was in the London Hospital under the care of Dr. Hughlings-Jackson, in which the appearance of the papilla was declared by an authority so decisive as that of Mr. Couper to be that of a "choked disc." No tumour, meningitis, or mechanism for "choking" was, however, discovered within the cranium. But the trunk of the nerve presented changes very similar to those in the case just mentioned, not, however, more intense at its posterior portion. Similar changes were found throughout the brain by Dr. Sutton, and it can hardly be doubted, taking the symptoms and anatomical changes together, that a condition, which must be regarded as "chronic irritation or inflammation" affecting the brain, had passed down the optic nerves and appeared as an intense papillitis, with signs of strangulation, due to the compression of the vessels within the papilla. A similar case has been recorded by Stephen Mackenzie.[1]

It seems from these facts that (1) a descending neuritis cannot be excluded from an examination of a small portion of the trunk of the nerve, and (2) that a change in the nerve revealing itself as a very slight deviation from the normal, may serve to convey a condition of irritation to the eye sufficient to light up considerable papillitis.

The frequency with which evidence of descending neuritis may be traced is confirmed by the observations of S. Mackenzie,[2]

[1] "Brain," July, 1879, p. 269.

[2] Loc. cit. and "Trans. Ophth. Soc.," vol. i. p. 94.

Brailey,[1] Edmunds and Lawford,[2] Poncet,[3] and others. Mackenzie has also pointed out that on no other theory than that of an inflammation travelling down the nerve tissue can we explain a unilateral neuritis on the side opposite to a cerebral tumour.

It has been maintained by Kuhnt[4] that the descent of inflammation from the brain to the eye is by the perivascular sheaths of the vessels, which are, he states, continuous with the pia mater of the brain, and Gayet[5] would ascribe a share also to the sheaths of the posterior ciliary vessels. The evidence of inflammation away from the vessels prevents us, however, regarding them as the exclusive agents, but pathological evidence of their participation in the transmission of the inflammation has been also brought forward by Edmunds and Brailey.[6]

It has been pointed out that the sheaths of the nerve, inner and outer, often present considerable changes, which make it probable that the inflammation passes along them to the eye. That it may do so independently of distension of the sheath is shown by two cases of optic neuritis and meningitis due to fracture of the skull, recently recorded by Edmunds,[7] in which the space between the sheaths of the nerve was occupied by "a dense mass of inflammatory products."

These facts suggest the following conclusions regarding the production of papillitis in intra-cranial disease:—

That in cases of cerebral tumour evidence of descending inflammation may be traced in sheath or nerve, much more commonly than current statements suggest, while in cases of meningitis the evidence of such descending inflammation is almost invariable.

That the resulting papillitis may be, and remain, slight,

[1] "Trans. Ophth. Soc.," vol. i. p. 111.
[2] Ibid., p. 112, and *loc. cit.* at p. 82.
[3] Disc. at the International Med. Congress, 1881.
[4] In a communication to the International Medical Congress at Amsterdam ("Ann. d'Oculist.," vol. lxxxii. 1879, p. 180).
[5] Ibid., p. 181.
[6] "Ophth. Hosp. Rep.," vol. x. p. 138.
[7] "St. Thos. Hosp. Rep.," vol. xi. 1881, p. 71; "Trans. Ophth. Soc.," vol. iii. p. 140.

or may become intense and present the appearances of mechanical congestion. The causes of this difference we do not yet know.

That such mechanical congestion does not, as a rule, result from compression of the vessels in or just behind the sclerotic ring, but always, when intense, from compression by inflammatory products in the substance of the papilla. It must not be forgotten that an increase in the size of vessels may be of reflex vaso-motor origin as in all inflamed parts.

That while slow increase of intra-cranial pressure has no effect on the retinal vessels, a sudden increase hinders the escape of blood from the eye for a time, and may intensify a papillitis originating in another way.

That distension of the sheath of the nerve alone is probably insufficient to cause papillitis by its mechanical effect, but may perhaps intensify the process otherwise set up, especially if the fluid possesses an irritative quality, and if (as Schmidt-Rimpler asserts and Leber denies) it can find its way into the lymphatic spaces of the optic disc.[1]

There being thus little evidence that a mechanical impediment to the return of blood from the eye—induced either by intra-cranial pressure, by distension of the optic sheath, or by the pressure of the sclerotic ring—ever plays any considerable part in the production of optic neuritis, the use of the term "choked disc" or "stauungs-papille," as indicative of a supposed mechanism, is to be deprecated in our present state of knowledge. The occurrence of a process of strangulation is not denied; it is often conspicuous enough, but it is produced in the inflamed papilla and not behind the eye, and occurs in all cases of a certain intensity.

In this outline of the facts regarding the origin of optic neuritis, the hypothesis that the mechanism is a reflex vaso-motor influence has been necessarily unnoticed, because the known facts have no bearing upon it and give it no support. It presupposes a special reflex relation not known to exist

[1] The latest theory of Leber, viz., that papillitis is an extension of inflammation from the periphery of the nerve at the anterior extremity of the sheath, is not supported by any anatomical evidence.

and a mechanism for the production of inflammation the efficiency of which is equally unknown.[1]

Varieties.—The chief varieties which have been usually insisted on are those distinguished by v. Graefe as "descending neuritis" and the "choked disc." The facts already mentioned make it more than doubtful whether the pathological basis of the distinction is correct, and it is generally admitted that the supposed distinctions cannot be relied upon. The aspect of the disc varies very much in the same case at different times; at one time the characters may be those supposed to be indicative of a descending neuritis, and at another time those ascribed to the "choked disc." But the appearance in different cases also frequently continues different throughout their whole course. These characters are so various, and the intermediate forms are so numerous, that it is exceedingly difficult to separate any varieties as special "forms." Some cases certainly present throughout characters which are regarded as those of descending neuritis—especially slightness of swelling, a tendency for the changes to be most intense in the peripheral part of the papilla, leaving the centre little affected, absence of hæmorrhages, the presence of white spots, isolated or about the vessels, and a striation depending rather on conspicuousness of nerve fibres than on vascularity. These changes are seen, for instance, in Pl. III. 3 and 5, and also of a wider extent and greater intensity in Pl. VI. 2. On the other hand, great swelling, with vascularity and distended veins, such as is seen in Pl. I. 6, III. 4, and still more in Pl. VI. 1, characterizes other forms. But in the case whose disc is shown in Fig. 16, descending neuritis presented the characters of

[1] A fuller consideration of the theory, and the arguments against it, will be found in some remarks I made in the discussion on optic neuritis at the Ophthalmological Society, March 10, 1881 ("Transactions," vol. i. p. 105). Similar arguments were brought forward by Leber at the discussion at the International Congress. The reflex theory has been revived by Loring ("New York Med. Journ.," June, 1882) in special connection with the fifth nerve, but still as a pure theory, which, while unsupported by facts (and even opposed by them), clearly merits detailed discussion.

the choked disc, while the changes in Pl. V. 1 and 2, 3 and 4, slight as they are, were in each case associated with the symptoms of intra-cranial tumour.

Until we know more of the relation between pathological process and ophthalmoscopic appearance, it seems far better to found varieties purely on clinical characters. Of varieties so founded the following have seemed to me the most marked.

1. *Slight Papillitis*, including the condition described above as congestion with œdema, in which the changes are so slight as to dim, but not obscure, the edge of the disc on indirect examination, although it may be invisible, wholly or in part, to direct examination (Pl. I. 3, 4, III. 3, 5, V. 1, 2, 3, 4.

2. *Moderate Papillitis.*—Obscuration of the edge of the disc, or of the affected portion, complete, even to indirect examination; swelling moderate, commonly reddish; veins natural or large; sometimes white tissue about the vessels, close to them or extending for some distance on the disc (Pl. I. 5, 6, III. 4, IV. 1, 3, V. 5, 6, VI. 2).

3. *Intense Papillitis.*—Great swelling; veins at first large and arteries small; many hæmorrhages; retina often involved by direct damage or by hæmorrhages. Always succeeds a slighter stage in which the evidence of strangulation may be at first little marked (Pl. VI. 1, VIII. 1).

The forms in which the changes involve the adjacent retina are often termed "neuro-retinitis circumscripta;" and such widespread change as is presented in Pl. VIII. 1, although originating in the papilla, merits such a designation. But in most cases, even in such as Pl. VI. 1, the retina is only affected adjacent to the papilla, or elsewhere is merely the seat of extravasations; and since there is no general inflammation of the retina, the term "retinitis" seems unnecessary.

Retro-ocular Neuritis.—The change known as such—an interstitial inflammation of the nerve—is a mixed condition of inflammation and atrophy, revealed in the disc, if revealed at all, by the signs of simple congestion, rarely those of slight

papillitis, and soon passing on to atrophy with narrowed vessels. Little is known of the exact anatomical changes in this form, except in the variety which has been termed *axial neuritis* (Förster), in which chronic inflammation occupies the axis of the nerve, and causes a central scotoma. It will be described further in the section on "Atrophy."

Retro-ocular Perineuritis is a condition of chronic inflammation of the sheath of the nerve leading to thickening of its tissues, and purulent infiltration among the trabeculæ. The nerve may suffer from compression, or from a state of interstitial neuritis which may spread to it from the sheath. It has been found in periostitis of the orbit (Horner), and in thickening of the cranial bones constricting the optic nerve (Michel). It causes papillitis in some, perhaps in all cases, but this does not necessarily assume the appearance described on p. 51 as "perineuritis."

Diagnosis.—The diagnosis of optic neuritis is often easy, but sometimes presents great difficulty. Of all its signs that which first attracts attention as the most conspicuous feature—the increased redness—is of least value, except in conjunction with other characters. As already more than once stated, the redness of a disc free from neuritis may nearly equal that of the adjacent choroid. The signs which are of greatest diagnostic value are (1) obscuration of the edge of the disc and (2) swelling. These, in conjunction with increased redness, or change of colour to a tint not normally seen (such as the peculiar lilac-grey so often presented), constitute the characteristic symptoms. The obscuration of the edge is especially significant. It indicates undue opacity of the tissue (layer of optic nerve fibres) in front of the edge. Most of the nerve fibres pass along the course of the great vessels, above and below the disc, and they often normally obscure the edge of the disc slightly in these situations. Sometimes they are densely packed, also, on the nasal side, especially when the central cup is very large, and a slight obscuration is produced there also; but in these cases, as a rule, the large size of the physiological cup indicates the close arrangement

of the fibres, the obscuration is slight and occurs in the normal situations, and the edge of the disc is elsewhere quite sharp. In these cases another character may occasionally be observed in a slight degree, which, in more intense form, is conspicuous in neuritis—the radiating striation at the edges of the disc. Normally this is seen where the nerve fibres are most closely aggregated, especially above and below; in morbid states it is to be observed all round the disc, although most intense where the nerve fibres are grouped, and it is then due not merely to pale lines (from swollen fibres with increased opacity), but in part, also, to red lines, fine vessels lying between the fibres.

The second indication of neuritis is the existence of distinct swelling. The prominence of one object in the fundus above the level of an adjacent object—*e. g.*, of a vessel on the edge of the physiological cup above a vessel at its bottom—is appreciated in the direct method of examination by moving the head of the observer from side to side, or up and down, as far as possible without losing sight of the objects. Their relative position undergoes an appreciable alteration proportioned to the difference in level, and is easily recognized. By the indirect method of examination the same result may be obtained by a lateral or vertical movement of the lens, which produces the same effect as a corresponding movement of the observer's head (the "parallactic test" of Liebreich). With the binocular ophthalmoscope these measures are unnecessary, the difference of level being apparent just as with the stereoscope. When the difference of the level of two objects is very great, as, for instance, in extensive swelling of the disc, a convex lens behind the mirror may be necessary before a clear view of the top of the swelling is obtained, the refraction of the eye being normal and the fundus visible without a lens. The difference between the strength of the convex lenses required to just render objects indistinct on the level of the retina and on the apex of the swelling, furnishes a measure of the height of the swelling.

Normally the surface of the papilla is a little anterior to the plane of the retina, hence the term "papilla." The amount of

this prominence varies in different cases. It is always greater where the nerve fibres are chiefly aggregated in the proximity of the retinal vessels, above and below, so that a transverse section through the disc may show scarcely any appreciable prominence, while a vertical section may present distinct prominence. The more closely the nerve fibres are aggregated in one part of the circumference of the nerve, the greater is the prominence. Occasionally, but not often in a normal eye, it is sufficient to be readily appreciable by the movement of the head in direct examination. As a rule, a prominence which is readily recognized is pathological. In morbid states, every degree of elevation may be met with.

The Diagnosis of the Cause of Papillitis.—The first question which presents itself in a given case is—Is the neuritis due to intra-cranial disease or to some other cause? The answer to this must, of course, depend on the presence or absence of indications of disease of the brain, or of such disease of the general system as is known to be accompanied by optic neuritis. The ophthalmoscopic characters of the neuritis will lead us a little way, but not far. A high degree of neuritis, with intense strangulation (such as the discs shown in Pl. VI. 1 and VIII. 1), is seldom met with except in cases of cerebral tumour and some forms of primary neuritis. The slighter degree of neuritis not uncommon in cerebral tumour, chronic meningitis, and other intra-cranial diseases, and the neuritis which occurs in Bright's disease, lead poisoning, &c., may resemble one another very closely. The neuritis of Bright's disease sometimes presents white spots in and close to the disc, but the same appearance may be, and often is, seen in the neuritis of intra-cranial disease. (Fig. 7.) White spots in the retina away from the disc, with papillitis of a slight degree, and presenting no evidence of a preceding more intense affection, is very suggestive of renal neuritis. The small cloudy spots seen, for instance, in Pl. IX. 2 (near the left edge of the figure), are of more significance than the minute white spots near the macula, such as are shown in Pl. IX. 3, although the latter are suggestive of renal disease when they occur with

a papillitis of slight degree and recent origin. Succeeding neuritis, or accompanying a neuritis which is subsiding, they are of much less significance, being often the relics of the mischief caused by simple inflammation; and how closely these may simulate the appearance of a renal retinitis is shown by Fig. 6 and Pl. VIII. 2. Although an appearance of so striking an aspect is very rare, a few white spots near the macula lutea are very commonly left by neuritis—such as are seen in Pl. VI. 1 and 3. The signs of a previous neuritis of considerable intensity—a prominent mass of tissue in front of the disc such as is seen there in Fig. 3, or a "filled-in" disc with evident compression of vessels, as in Pl. VIII. 2—rarely coincide with a similar appearance in renal retinitis, although such a coincidence is seen in Pl. IX. 4. In such a case as is there figured the diagnosis of the cause of the neuritis could scarcely be made by the ophthalmoscope alone. But attention must always be paid to the degree of the present inflammation, or the evidence of its degree in the past afforded by the amount of new tissue formed.

It is upon the independent signs of one or the other causal condition that the diagnosis must chiefly turn. In referring neuritis to cerebral mischief, it must not be forgotten that, on the one hand, optic neuritis due to a cerebral tumour may be accompanied for a time by no signs of intra-cranial disease, and, on the other hand, that an optic neuritis due to a general disease may be accompanied by symptoms suggestive of cerebral disturbance, especially headache, vomiting, and even, in some cases, convulsions. Striking instances of the former were afforded by two children whom I saw at the same time in the Great Ormond Street Hospital. One was a boy under the care of Dr. Barlow, with a tubercular growth within the right eyeball, and well-marked neuritis to be seen in the left eye (Pl. III. 4). The only other symptom suggestive of intra-cranial mischief was an occasional attack of vomiting during many months that he remained under observation. The neuritis was of the character highly suggestive of intra-cranial tumour, but the possibility that the mischief in one

eye might have caused the neuritis in the other, suggested extirpation of the eye which was the seat of the tumour. It had, however, no influence; and when the boy died, about a year after, scrofulous cerebral tumours were found. The other case (under the care of Dr. Gee) was a child aged nine years, who was admitted having had occasional attacks of headache and vomiting. During the intervals she seemed perfectly well. No symptoms referable to the nervous system could be detected. She had, however, double optic neuritis. Gradually unsteadiness of gait showed itself, and increased until she was unable to stand, and she ultimately presented all the symptoms of cerebellar tumour. Such facts show that the suspicion of intra-cranial disease in cases of optic neuritis can only be discarded after long observation, if indeed it can ever be given up until some other cause presents itself. This is especially the case when the neuritis is chronic: very acute neuritis is nearly always accompanied by symptoms indicative of the disease causing it. Tubercular tumours of the brain frequently cease to trouble the patient or his optic nerves, and the cessation is permanent.

On the other hand, neuritis due to general disease may be accompanied by symptoms suggestive of cerebral mischief. The disc shown in Pl. IX. 3 is that of a man who complained of almost constant severe headache and occasional attacks of sickness. The ophthalmoscope showed well-marked neuritis, moderate in degree, and on first inspection no retinal disturbance was detected. It was thought, for the moment, to be a case of cerebral tumour. On looking more carefully by the direct method, however, near the macula lutea were seen a number of minute white spots inconsistent with the slight degree of neuritis. The urine was at once examined, and found to be loaded with albumen, and on further examination hypertrophy of the heart and a hard pulse were found, with some signs of uræmic mischief. He died of uræmia not long after. The history of the case shown at Pl. IX. 2 is similar, except that the evidence of cerebral disturbance here was mental change, not headache. Another case impressed itself very strongly upon me many years

ago, when, as a resident in University College Hospital, I was first working with the ophthalmoscope. A man was admitted with convulsions, and comatose. An examination of the eyes showed double optic neuritis, and a diagnosis of cerebral tumour was at once ventured on. The patient died in a few hours, and the necropsy revealed contracted kidneys and a normal brain. A mistake of this kind is easily made, especially if the examination is confined to the indirect method; but I think that the mistake may generally be avoided by the direct method of examination, which has, in all cases I have since seen, disclosed slight retinal alteration inconsistent with the form of the neuritis. Examination of the urine should, of course, never be neglected.

Headache and vomiting are, then, the signs of least value as indications of an intra-cranial cause of neuritis. Convulsion is also of little value unless it is of a form which indicates local brain disease, *i.e.*, local in distribution or in commencement.

In all obscure cases, search must be made for any other cause of optic neuritis, especially lead poisoning. In cases of lead poisoning renal disease is very frequent, and that cause for neuritis must be excluded before the affection can be referred to plumbism. In these cases also doubt may be felt as to whether the mischief is not due to cerebral disease, because lead poisoning is sometimes accompanied with two forms of cerebral disturbance—delirium and convulsion. In the case presenting the neuritis shown in Pl. VII. 6 there was extreme cerebral disturbance, apparently the consequence of the lead poisoning; and, on the other hand, I have lately had under my care several cases in which recurring convulsions, precisely like those of idiopathic epilepsy, were due to the same cause.

One other fact must be mentioned in connection with the diagnosis of the cause of optic neuritis. In many cases in which slight neuritis of chronic course is associated with symptoms which would scarcely suggest the existence of disease such as would cause neuritis, hypermetropia exists. This combination may be noted, for instance, in chlorosis (as in the case figured in Pl. VII. 5), in epilepsy, apparently

idiopathic, and other slight symptoms of cerebral disturbance. It is doubtful, in the present state of our knowledge, what share is to be attributed to the hypermetropia in the production of the neuritis, and from the commonness of hypermetropia the coincidence may have been accidental, but the fact deserves notice.

Prognosis.—The prognosis in optic neuritis is necessarily a source of considerable anxiety. In few cases can it be said that vision is not in danger of impairment and even of loss. The prognosis must be formed by a careful study of the conditions on which impairment of sight depends, as stated on p. 72. The prospect is better in the slighter degrees of papillitis, and better in proportion to chronicity of course, and dependence on causes which can be treated. It is worse when there is reason to believe that there is much retro-ocular mischief; worse in proportion to the evidence the ophthalmoscope affords of a process of compression going on in the disc; worse in proportion to the intensity of the changes; and worse in the loss of sight which comes on during the recession of the inflammation than in that which comes on during its height.

The cause of the optic neuritis must influence our prognosis more than any other condition. It is better in syphilitic than in scrofulous cases, and better in these than in cases of disease of other forms. Even in syphilitic mischief, however, the prognosis must be guarded if the intra-ocular changes are considerable. It is not probable that the optic neuritis is, itself, syphilitic in nature. Its subsidence depends rather on the subsidence of the syphilitic intra-cranial disease, than on the influence of the remedy on the intra-ocular process, and it is not uncommon to have considerable failure of sight during the subsidence of the neuritis in such cases. Fortunately when the subsidence of the neuritis has ceased, there is a greater tendency to improvement of vision, and this may be considerable in degree (see "Consecutive Atrophy").

reatment.—Very little can be done for the direct treat-

ment of optic neuritis. The treatment is that of the intra-cranial mischief, or general disease, which is its cause. Beyond this, local measures, leeches and the like, are little likely to influence the progress of the disease. The puncture of the distended nerve-sheath has been advocated by De Wecker, and performed by him and by Mr. Power, and recently in a number of cases by Mr. Brudenell Carter and Mr. Bickerton.[1] It is based on the theory that the distension of the sheath is the cause of the intra-ocular neuritis, a theory which, it has been seen, cannot yet be considered as proved. Improvement has followed the operation in a few of the cases, but it must be tried in a much larger number of cases before a decisive opinion can be formed.

During neuritis the eyes should be used as little as may be, and such conditions as intensify intra-ocular congestion should be avoided, *e.g.*, exposure to cold, and all causes of mechanical congestion, straining, cough, &c. Ice to the forehead has been recommended by Pflüger.

Optic neuritis is so frequently associated with syphilitic disease of the brain and its membranes, and the evidence which may seem to exclude the suspicion of syphilis is so often misleading, that the administration of iodide of potassium should be a rule in almost all cases in which the age of the patient is such that acquired syphilis is possible. Iodide, in large doses, secures a more prompt improvement than mercury, and does no harm if the disease is not syphilitic in nature. Additional benefit may, however, result from the subsequent use of mercury. The completeness of recovery depends on the promptness with which the progress of the disease can be checked. Even in syphilitic cases it must be remembered that, the intra-ocular neuritis being probably not syphilitic in nature, although the consequence of syphilitic brain disease, the remedy employed does not influence the inflammatory products in the papilla, as it does the disease in the brain. As it has just been stated, in many cases of syphilitic disease of the brain with optic neuritis, in which the cerebral symptoms have cleared, and the neuritis has subsided under

[1] "Oph. Rev.," 1888, vol. vii. p. 300.

appropriate treatment, sight has become damaged during the subsidence of the neuritis, apparently very much as it would have done had the cerebral disease not been syphilitic in nature. It is the recession of the cerebral trouble which permits the recession of the neuritis, and the ocular damage bears, in most cases, a direct proportion to its duration. Hughlings-Jackson believes that iodide of potassium is sometimes useful when there is no syphilis. Iodoform, internally and externally, has been advocated by Landesberg.

Where the disease is not syphilitic it is often scrofulous, and here also great good can be done by appropriate—especially tonic—treatment. Commencing neuritis may subside entirely and leave no trace, under the influence of such treatment. But unfortunately we are able to influence such disease much more slowly than we can influence syphilitic disease, and if neuritis be already well developed, it is rarely that loss of sight can be prevented.

B.—*MORBID STATES OF THE OPTIC DISC CHARACTERIZED USUALLY BY LESSENED VASCULARITY AND SIGNS OF WASTING. ATROPHY OF THE OPTIC NERVE.*

Under many circumstances the fibres of the optic nerves undergo wasting or degeneration. This occurs when the eye has been greatly damaged by any cause, and possibly when complete opacity has rendered the cornea or lens, for a long time, impermeable to rays of light. It has been seen to occur as a consequence of the inflammation of the intra-ocular end of the nerve, or of its whole trunk; the wasting thus produced is termed "consecutive," "post-papillitic," or "post-neuritic atrophy." In other cases the wasting is preceded by no visible inflammatory disturbance, and such are termed "simple atrophy." Nevertheless, in rare cases, an atrophy is preceded by the signs of simple congestion of the disc, and such cases may be termed "congestive atrophy." It is probable that the pathological condition of the optic nerve in this form is really a chronic inflammation, partial or diffuse, of which the intra-ocular signs of congestion, &c., are the indication, but

it is convenient, for clinical reasons, to consider it among the forms of atrophy. Lastly, atrophy may succeed choroiditis and retinal disease.

Atrophy, not consequent on any obvious ocular change, was found by Vulpian in about 4 per cent. (19 out of 500) autopsies on old persons at the Salpétrière. In an equal number (21) there was atrophy consequent on an ocular disease.[1]

Characters.—The nutrition of the nerve fibres, and that of the capillary vessels which confer on the disc its normal rosy tint, are so associated that atrophy of the fibres is accompanied in nearly all cases by an atrophy of the capillaries, and the pallor thus produced constitutes the most salient sign of the atrophy of the nerve. The atrophied nerve commonly shrinks, and occupies less bulk than the normal nerve. This is not attended by any diminution in the size of the optic disc, since the latter is determined by the size of the sclerotic opening. The shrinking is indicated by a slight recession or "excavation" of the disc. In some cases there is a diminution in size of the retinal vessels, but this is an inconstant character. These signs will be considered in detail.

Pallor.—The vascularity of the optic nerve, as has been before pointed out, is estimated by the tint of its intra-ocular termination, the "optic disc." In judging of the colour of the disc it is important to examine it with a weak illumination, and by the direct method, in order to let as little light as possible be reflected. In a strong light a faintly-tinted object will appear white.[2] Hence the importance, to recognize a slight coloration, of employing a weak illumination. The ophthalmoscope of Helmholtz, consisting of plates of

[1] Table given by Galezowski, "Sur les Atrophies de la Papille du Nerf Optique." "Journal d'Ophtalmologie," Jan., Feb., and March, 1872.

[2] With very intense illumination, even a strongly-tinted object will appear white. This is because all objects reflect some of all rays, and absorb none entirely. If the waves impinging be sufficiently numerous—*i.e.*, the light very intense—so many waves of all lengths are reflected that the object appears white, the waves of the length chiefly reflected being no longer preponderant, although they become preponderant on weakening the light.

thin glass, is especially useful for this purpose. A plane mirror may be employed instead. If this is not available, the light of the illuminating lamp should be turned low.

It is as essential to be aware of the normal variations in colour for the estimation of a pathological pallor of the optic disc, as it is for the recognition of congestion. The variations on the negative side are not, perhaps, so considerable as are those on the positive side, but they are sufficient to render familiarity with the appearance of the normal disc essential to prevent mistakes in estimating the slighter degrees of atrophy. As a rule, the disc becomes paler as life advances, and a slight grey tint becomes mingled with the red, but the latter is still perceptible. The physiological cup, if slight, is often indistinct late in life. Thus, a tint which is normal in the old, would be suggestive of atrophy in the young. Again, when the general fundus is unusually dark, the disc will seem to be abnormally pale, simply as an effect of contrast. In anæmia, also, the disc may become paler, but the change of tint from this cause is not considerable, and is insignificant in comparison with the normal variations in colour of the disc. It never constitutes an element of difficulty in the recognition of atrophy.

When a pathological pallor of the disc is pronounced, it extends over the whole area of the disc, but commencing pallor may be most marked in that part of the disc which is normally palest, *i.e.*, the temporal side, where the nerve fibres are least numerous. The change in this part, however, is only of significance in individuals in whom the "physiological cup" is small, and the temporal half of the disc normally possesses a distinctly vascular tint. In a great number of cases, in which the physiological excavation is large, and slopes gradually to the sclerotic ring on the temporal side, this portion of the disc may be normally almost as pale as in atrophy. The part on which attention should be chiefly fixed is, therefore, that which normally possesses considerable vascularity, the nasal portion. The tint may be observed to become gradually paler, the red sometimes simply fading, and leaving a white colour in its place; in other cases a grey

becomes mingled with the red, and gradually preponderates as the red tint fades, and ultimately a pure grey is left. If the examination is made with daylight, the tint is often a greenish-grey. These two varieties constitute in their extreme forms the white and grey forms of atrophy respectively. Intermediate forms are often seen, and to the direct method of examination some grey tint may always be distinguished, even in the discs which appear of tendinous or chalky whiteness to the indirect method of examination. This grey mottling tends to increase as time goes on. The slight grey tint in "white atrophy" is similar to that normally seen at the bottom of the physiological cup. This tint is, however, scattered over the disc, and the central cup is often distinguishably whiter or greyer than the rest.

The aspect of the disc, whether white or grey, is not definitely related to the form or cause of the atrophy, and hence it is undesirable to employ it as a basis for classification.

The atrophy leaves the edge of the disc very distinct and sharp. The sclerotic ring is much more clear than it is normally, but it may not at first be recognized by the indirect method, as it is not differentiated from the white surface, as it is from the rosy tint of the normal disc. The sharpness of the edge is due, not only to its clearness, but also to the fact that the choroid preserves its normal characters to the margin, and gives to the clear outline a peculiar sharp-cut aspect, which is the characteristic of "simple atrophy." Pigmentary deposits on the edge of the disc are, like the edge itself, abnormally distinct.

Excavation.—In simple atrophy of the nerve, the surface of the disc is depressed in proportion to the wasting of the nerve-trunk. This varies, however, in the different forms of atrophy, because the wasting of the nerve fibres is, in some forms, combined with wasting of the connective elements, and a great shrinking of the nerve in size, while in other cases the wasting of the fibres is accompanied with an overgrowth of connective tissue, which may to some extent compensate for the shrinking due to the atrophy of the nerve elements, and may even prevent any diminution in bulk of the nerve.

Thus, in some cases, the depression of the disc is considerable, and in others it is slight or absent. Its special character is that it affects the whole disc, and commences at the sclerotic ring. It may often be recognized by the change of level of the retinal vessels at the spot, most distinct on lateral movement of the observer's head. Normally, it will be remembered, the depression of the centre of the disc never begins at the sclerotic ring, except that in some cases of large normal cups it may commence at the ring on the temporal side. Above, below, and at the nasal side—*i.e.*, in the position of the large vessels—the normal excavation never commences at the ring, within which there is always a zone of nerve tissue, commonly the most prominent portion of the disc. Hence the change of level of the large vessels at the ring becomes an important sign of the atrophic excavation. The size and form of the resulting excavation depend on two things—the amount of shrinking of the nerve, and the size and form of the normal cup. The wasting of the edge of the cup tends to lessen the steepness of the side or sides, and to give its form a funnel shape.

The mottling of the lamina cribrosa may become very distinct at the bottom of the excavation, and this in some cases, it is said, in which before the atrophy no physiological depression existed. Where the normal cup was large, the excavation may reveal the lamina cribrosa in almost the whole extent of the disc, the grey mottling corresponding to the bundles of degenerated nerve fibres, the white intervals to the meshes of the lamina.

It is believed that some share in the excavation is due to the atrophy of the small vessels, which conferred on the normal disc a certain amount of turgescence. De Wecker suggests that as the nerve has its consistence lessened, the normal intra-ocular pressure may assist in producing the excavation.

It has been said that the more connective tissue is developed in the atrophied nerve, the slighter is the shrinking of the trunk. This is especially the case in the grey atrophy, in which the nerve may retain its normal size. The de-

pression in the disc may be less in these cases than in the whiter form of simple atrophy, but it is not, as has been said, absent, and it is often considerable. Among the remains of the diverging nerve fibres, there is little connective tissue developed, and the wasting of the fibres here is compensated for to a much less extent than in the trunk of the nerve.

The Retinal Vessels.—In some cases of simple atrophy of the optic nerve the retinal vessels become reduced in size, in others they do not. In the grey atrophy, as a rule, the vessels undergo little or no change, but they are occasionally narrowed. In simple white atrophy they present no alteration in some cases; in others, the arteries gradually become smaller, the veins undergoing little diminution. After a time the veins also may shrink. They are reduced in size in cases in which there is a retro-ocular neuritic process, but this, without evidence of neuritis in the disc, cannot be regarded as the cause of their shrinking in all cases. Their atrophy seems sometimes to be part of the atrophy of the nerve-fibre and ganglion-cell layers of the retina, which is usually associated with atrophy of the nerve. Why they should shrink in some cases and not in others is at present unexplained.

Initial Signs of Congestion.—In describing simple congestion of the disc, it was pointed out that it may terminate in atrophy. The disc has, at first, a dull-red tint, with a soft-looking surface, the redness being uniformly distributed over it. The edges of the disc are less sharply defined than in health; they are visible, but are softened. It is this uniform distribution of the tint, and softness of the edge, which give to the disc its special character. The congestion may persist for a long time, but commonly, as time goes on, the disc slowly becomes paler, and ultimately a condition of greyish-white atrophy is reached. Occasionally the disc presents at first, for a short time, a slight degree of œdema as well as congestion, shown by slight swelling. The pathological process, in many cases of atrophy, seems to be of the nature of a chronic inflammation. It is readily intelligible that in some cases the signs of slight inflammation should be

visible in the disc during the early stage. The cases in which it is met with are especially those which result from injury and from toxic causes. This state of chronic inflammation behind the eye, retro-ocular neuritis, may be diffuse and affect the whole nerve, or partial and involve only a segment of the nerve (segmental neuritis), or its central portion (axial neuritis). The vessels often present much earlier and more considerable narrowing than in simple atrophy, and in the disc around them much white tissue becomes developed. It is to be noted, however, that in some conditions of undoubted retro-ocular neuritis, there may be no signs of inflammation or congestion of the disc, but only that of simple atrophy, and hence it is convenient to consider this form in the present section. The mischief is commonly at some distance behind the eye.

Atrophy after Intra-Ocular Neuritis; "Consecutive Atrophy," or "Papillitic Atrophy."—The newly-formed inflammatory tissue-elements of papillitis are in part removed, and in part transformed into connective tissue, which gradually shrinks. The pale swelling left by the inflammation (Pl. II. 1, IV. 5, VI. 3), large in proportion to the intensity of the process, slowly subsides, until it is confined within the limits of the disc, and slowly reaches the level of the retina (Fig. 49). The soft edges which at first limit the pale swelling gradually become more sharply defined. The recession of the swelling

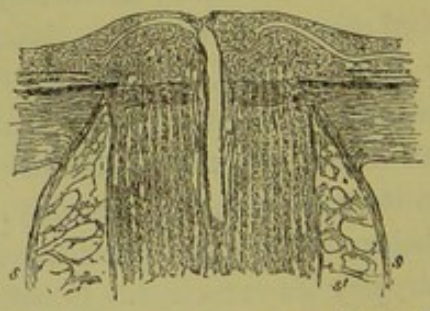


FIG. 49.—VERTICAL SECTION THROUGH THE OPTIC DISC IN A CASE OF POST-PAPILLITIC ATROPHY, DUE TO TUBERCLE OF THE CEREBELLUM.

The retinal layers are displaced, and the bundles of fibres in the optic nerve are separated. A vessel is seen divided longitudinally. Neither within nor behind the sclerotic ring is it compressed. Within the papilla, however, its branches are very narrow. (× 15.)

from the edge of the choroid often shows that the latter has been damaged, and has undergone irregular atrophy adjacent to the edge of the disc (Pl. II. 4, IV. 4), which thus has a more or less irregular outline. The substance of the disc has a "filled-in" look, from the new tissue within it (Pl. VIII. 2), and is commonly white, or rarely greyish in tint (Pl. II. 2, upper half). The vessels, whether previously narrowed or not, usually become narrowed by the contraction of this new tissue, and may be partly concealed by it at their origin, or in their course over the disc. The tissue along their walls is often distinctly whiter than the rest of the disc, and when the latter is grey the contrast between it and the perivascular tissue may be very marked (Pl. II. 2). Often white lines are to be traced along the narrowed vessels for some distance from the disc (Pl. II. 4). They are probably due to thickening of the outer coat, perhaps originating in the migration of white corpuscles along the perivascular sheaths (Fig. 11), and the transformation of these into connective-tissue elements. Ultimately, the contraction of the tissue may cause an excavation of the disc, even in the centre (Pl. II. 4, IV. 6), and there is only the adjacent choroidal disturbance and the narrowing of the vessels, to indicate the origin of the atrophy. The excavation rarely, however, becomes sufficient to reveal the lamina cribrosa. (Cf. Figs. 3 and 4, Pl. II.) The disc usually remains for a long time white to the indirect examination; sometimes its tint is slightly rosy. Ultimately, however,

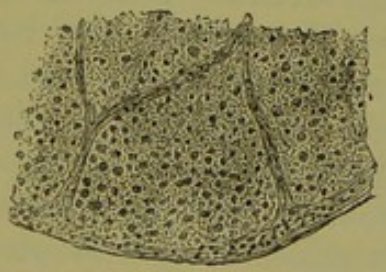


FIG. 50.—SECTION THROUGH THE OPTIC NERVE IN THE SAME CASE AS THE PRECEDING FIGURE.

The fasciculi of degenerated nerve fibres are infiltrated with nuclei, and cells of irregular shape. The septa between the bundles are a little thickened. × 100.)

it becomes distinctly greyish, especially on direct examination, and with feeble illumination. In some cases the inflammation may not have damaged the choroid, although causing destruction of the nerve fibres, and in such a case the edge of the disc may be sharply defined, and if, as is the case sometimes when the inflammation is moderate, the narrowing of the vessels is slight in degree, the appearance of the disc may resemble very closely the disc in simple atrophy, and be quite indistinguishable from that left by retro-ocular neuritis.

Choroiditic Atrophy.—The atrophy of the disc, which is often seen after choroido-retinitis, is sometimes white or grey and resembles primary atrophy; but sometimes it presents special features, being characterized by a peculiar reddish, or yellowish-red tint of disc, uniform in distribution, sometimes with slight blurring of its edges, and usually by a marked wasting of the retinal vessels, which may be diminished in number as well as in size.

The recognition of this variety of choroiditic atrophy is of considerable importance, because, unless the result of retinitis pigmentosa, it is almost always the consequence of syphilitic disease, acquired, or more frequently, inherited. It constitutes a sign of inherited syphilis of great importance. In most cases the disturbance of the retinal pigment is distinct and characteristic.

Causes.—Simple atrophy of the optic nerve may be a primary change, or may be secondary to some lesion, traumatic or other, which interferes with the structural integrity of the nerve. These two varieties may be distinguished as "primary" and "secondary" atrophy, and are especially characterized by the circumstance that in primary atrophy the loss of sight coincides in origin and progress with the visible atrophy, but in secondary atrophy the loss of sight occurs first, and the signs of nerve degeneration are not observed until a subsequent period. It is doubtful whether the two forms can be distinguished by the ultimate aspect of the disc.

It has been proposed to divide the primary atrophies into two classes, according as the process commences by degeneration

of the nerve elements, or by growth of the interstitial tissue, with secondary damage to the nerve fibres. The distinction has been especially insisted on by Charcot and by Abadie, on grounds of etiology, pathology, and symptoms. Our knowledge at present is scarcely sufficiently definite to make a sharp distinction generally useful, if indeed it is founded on a correct basis. The careful discussion of the subject by Duwez deserves perusal.[1]

Primary Atrophy often comes on without known causes. It is sometimes, however, distinctly hereditary, and one very remarkable form (carefully studied by Leber) affects all the males of a family soon after puberty. The atrophy is here really preceded by a slight neuritis, and its occurrence seems to be associated with a neuropathic type of family.[2] The male sex is, apart from this variety, more prone to optic nerve atrophy than the female. Seventy-five per cent. of all cases occur in men, and most cases occur in adults. A considerable number of the cases of primary atrophy are associated with spinal disease and are distinguished as "spinal atrophies." Cases of optic nerve atrophy, in which there are no symptoms of other affection of the nervous system, are usually classed as "simple progressive atrophy"—an inconvenient designation, since the cases of spinal atrophy are also progressive. The class probably includes several distinct forms which are not yet differentiated.

The group of "spinal atrophies" of the optic nerve is of great medical interest and practical importance. The most important is the atrophy which so often accompanies locomotor ataxy. This form is regarded as the most typical example of the "parenchymatous," *i.e.*, primarily neural form. It is usually a grey atrophy in ophthalmoscopic aspect, without diminution in the size of the vessels. A large number of primary atrophies are of this variety. The tabetic symptoms may be long delayed, and many such cases

[1] In the "Dictionnaire Encyclopédique des Sciences Med.," tom. xvi. pt. 1, p. 319.

[2] See also a paper on this subject by S. H. Habershon: "Trans. Ophth. Soc.," vol. viii. 1888, p. 190.

have been regarded as independent atrophy (see Part II., "Diseases of the Spinal Cord"). It has been indeed suggested by Charcot that almost all cases of primary atrophy are of this form, that the subjects of them, if they do not present spinal symptoms when seen, will do so at a future period. This is certainly incorrect. It is probable, from the facts observed by Uhthoff,[1] that not more than one-half of the cases of primary atrophy are associated with disease of the spinal cord.

A similar atrophy may be observed occasionally in general paralysis of the insane, and also, although rarely, in disseminated (insular) sclerosis, and in lateral sclerosis of the cord. The form which occurs in general paralysis is described by Clifford Allbutt as often preceded by distinct signs of congestion of the disc. This is doubted by many, and is certainly very often not to be observed, but in one or two cases I have seen marked congestion of the discs in general paralysis, although unable to follow them to the atrophic stage.

The pathology of the connection of the optic nerve atrophy and the spinal cord changes is still obscure. The fact that in locomotor ataxy the atrophy may reach an advanced degree when the change in the spinal cord is still in its earliest stage, and even when the latter is confined to the lowest part, makes it probable that the optic change is an associated and not a sequential lesion. At present this probability is not lessened by the discovery of J. Stilling[2] that some fibres of the optic nerve can be traced into the medulla oblongata as far as the inferior olivary body.

It must be remembered that the optic nerve is, developmentally, a direct prolongation of the central nervous system, and that, anatomically, it resembles the white matter of the brain and spinal cord. The importance of this relationship, in connection with the question of the independent origin of changes in the optic nerves and in the spinal cord, has been called attention to by Gunn.[3]

1 "Arch. f. Ophth.," vol. xxvi. 1881, pt. 1, p. 277.
2 "Centralblatt f. prakt. Augenheilk.," Dec. 1880, p. 377.
3 "Brit. Med. Journal," 1885, ii. p. 688.

The atrophies of the optic nerve which are not associated with spinal disease have been ascribed to various causes, the influence of some of which is uncertain. Such are: cold, sexual excess, menstrual disturbance, gastro-intestinal affections, migraine. With better reason they have been ascribed in rare instances to syphilis, diabetes, intermittent fever, and some acute specific diseases, and the facts regarding their relation to these will be considered in Part II. In a considerable proportion of the cases of primary atrophy unconnected with spinal disease, no adequate cause can be ascertained. Tobacco and bisulphide of carbon certainly, alcohol and lead possibly, cause amblyopia, and may cause partial atrophy, but this is usually preceded by signs of congestion or even inflammation, and there is reason to believe that, in the case of tobacco at least, the lesion is a neuritis in the axis of the nerve.

Primary atrophy usually affects both eyes, commonly one much more, and earlier than, the other, and in rare cases one only.

Secondary Atrophy results from lesion of the optic centres or fibres. A cortical lesion in the brain about the supra-marginal gyrus (Ferrier) may, there is reason to believe, entail loss of sight of the opposite eye. This, although the decussation at the chiasma is certainly in man incomplete, is explicable by Charcot's at present unproved theory of a complemental decussation at the corpora quadrigemina. A lesion outside the hinder part of the optic thalamus causes, according to this theory, loss of sight of the opposite eye and of the opposite half of the field of vision of the same side. It is probable that such damage does not for a long time cause atrophy of the disc. The case from which Pl. II. 5 is taken makes it probable that such atrophy after a time does ensue; and the same conclusion is suggested by a case recorded by Bernhardt.[1] Lesions of one optic tract causing bilateral symmetrical hemianopia, seldom produce distinct ophthalmoscopic changes. Some observers have described an ultimate slight pallor of the corresponding halves of the

[1] "Berl. kl. Wochenschrift," 1872, No. 30.

discs, but this is not often distinct. In one case of long duration, in which the hemianopia was complete and persistent, in the course of years the whole of the disc of the eye in which the area lost was on the temporal side (and therefore greatest), became perceptibly paler than the other, the tint of the two being at first equal. A similar slight pallor of the disc opposite to the cerebral lesion has been noted by others in cases of hemianopia of long duration.

Pressure on the chiasma or nerves at the base of the brain is a common cause of optic nerve atrophy without neuritis. In the case figured in Pl. II. 4, although there had been slight neuritis, the atrophy was probably due to this cause. The pressure may be that of tumours growing from any of the adjacent structures, exostoses from the bone, or aneurisms from the adjacent arteries. It not uncommonly results from internal hydrocephalus—the distended third ventricle compresses the chiasma directly, pressing first on the upper and posterior aspect, where, as Michel has shown, a depression may be thus produced.[1] Meningitis is another cause which, while commonly producing optic papillitis, if extending to the nerve, may, in rare cases, cause blindness and atrophy without intra-ocular inflammation, by pressure without inflammatory invasion, or it may cause blindness and atrophy out of proportion to neuritic mischief, and often after the inflammation of the papilla has subsided. It is probable that the local neuritis in these cases is often much more intense than is suggested by the degree of intra-ocular inflammation. It is said that obstruction, by embolism or thrombosis, of one middle meningeal artery, which supplies the dura mater around the optic foramen, may be followed by atrophy of that optic nerve. Tumours, exostoses, and meningitis may damage the nerves in front of the chiasma, and so affect the two eyes equally, or one to a much greater extent than the other, or one exclusively. The atrophy from these causes is white or grey.

[1] Compression and flattening of the chiasma from ventricular distension was noted by Cheselden in the last century. ("Phil. Trans.," No. 337, p. 281.)

Damage to the optic nerves causing atrophy may also occur in the optic foramen or in the course of the nerve through the orbit. Narrowing of the foramen by bony thickening, and rheumatic or syphilitic or traumatic mischief, producing pressure at the back of the orbit, close to the foramen, are not rare causes of atrophy. Blows on the head commonly produce atrophy by direct injury to the nerve, but it is probable that they occasionally cause, by the effect of the shock, a gradual degeneration. The ultimate atrophy which results from these causes is usually more or less distinctly grey in aspect, and the grey tint may be as marked as in the form supposed to be characteristic of spinal disease (see Pl. II. Fig. 3).

Mischief in the orbit may cause a process of "retro-ocular neuritis." This is assumed when transient signs of congestion are present in the disc, accompanied by constriction of vessels and the development of tissue adjacent to them; so that ultimately there is considerable narrowing of the retinal vessels, as in the atrophy which is consecutive to intra-ocular neuritis. Sometimes the signs of neuritis are more marked. The nerve may be thus damaged by the extension of inflammation to the orbit in erysipelas of the face.

Papillitic or consecutive atrophy results from intra-ocular neuritis, as already described.

Retinal and Choroiditic Atrophy.—Lastly, damage to the retina entails an atrophy of the optic nerve, which progresses, sometimes slowly, sometimes quickly, but is usually incomplete. Now and then atrophy of the optic nerve follows a cause which seems to act by giving a shock to the retina, that leaves no trace behind—*e.g.*, the complete amaurosis, which may accompany the onset of embolism of one branch of the retinal artery, and is usually temporary, may sometimes be permanent, even though all the other branches of the retinal artery are previous. Atrophy sometimes follows a blow on the eye, as in a case related by Laqueur, in which a blow caused complete amaurosis without visible changes in the fundus, and simple atrophy followed. Such cases are of medical interest on account of the light they throw on the action of some general causes. It is rarely that any con-

siderable degree of atrophy follows retinitis. Commonly, the cause of retinal atrophy is obvious on ophthalmoscopic examination, and the medical interest of this form is subordinate to that of the retinal change. After choroiditis the disc has often a yellowish-red tint, as already described (p. 110).

Anatomical Changes.—Atrophy of the optic nerve is never confined to the papilla; the changes are marked throughout the whole length of the nerve, and in primary atrophy are usually equally distributed. The size of the nerve varies very much; in some forms of primary atrophy it is markedly smaller than normal, somewhat translucent but scarcely grey, and under the microscope may present merely a wasting of all the structures of the nerve, fibres and connective elements, with, especially in recent cases, products of the degeneration of the nerve fibres, granules and globules of fat, compound granule cells, "corpora amylacea," and other products of degeneration of the nerve fibres. The position of the latter may at first be marked by rows of fatty particles. In other cases the nerve may be little diminished in size, but may present under the microscope a great increase in the interstitial connective tissue, fibres, and cells, with disappearance of the nerve tubules. Commonly the change is greater in the circumferential portions of the nerve than in the central.[1] Occasionally the reverse is the case. In atrophy from pressure on the nerve, its size is usually greatly reduced, and the increase of connective tissue is very considerable.

In primary grey atrophy the nerve trunk is usually little reduced in size, and is grey and gelatinous in appearance. Microscopically, it presents an increase in the connective tissue trabeculæ, and an atrophy of the nerve fibres. The medullary sheath first disappears, and afterwards the axis cylinder. It is said that the nerve fibres may be reduced to fine fibrous threads. Products of myelin degeneration may be found in the earlier stages. Sometimes the change is

[1] Leber: "Arch. f. Ophth.," xiv. p. 182.

peculiar; there developes round the vessels a peculiar gelatinous-looking tissue containing a few nuclei and indistinct concentric fibrillation. The normal arrangement of the trabeculæ disappears, and a section of the nerve (Fig. 51) shows islets and tracts of this tissue, in the centre of each of which a vessel can be traced. They may occupy at least half the area of the section. Between them lie the fasciculi of degenerated nerve fibres with little increase in their interstitial tissue. In the case figured, the atrophy was confined to one optic nerve, and its cause was obscure. The same histological condition may be present in the grey atrophy of locomotor ataxy.[1] In other cases of grey degeneration (according to Leber's observations) the change may be more uniformly distributed through the fasciculi. The degeneration is sometimes found in certain areas much more intensely than elsewhere. In a case of locomotor ataxy in which sight was not known to be impaired, I found only a great increase of tissue, consisting of nuclei and fibres, at the nodal points of the trabeculæ, and a little gelatinous-

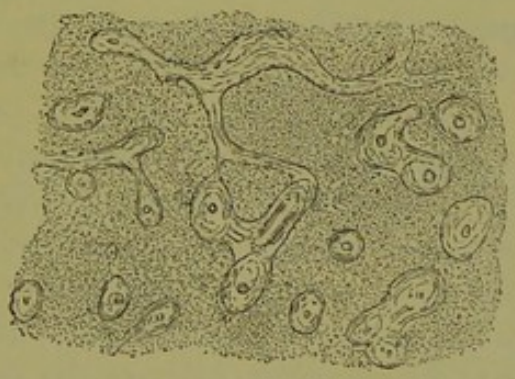


FIG. 51.—GREY ATROPHY OF OPTIC NERVE: TRANSVERSE SECTION, MIDWAY BETWEEN THE EYEBALL AND THE OPTIC FORAMEN.

The trunk of the nerve was grey, and gelatinous in aspect, and was not diminished in size. The other optic nerve was healthy. The nerve fibres are completely degenerated, a granular tissue representing them. The normal trabeculæ have disappeared, and through the section of the nerve are scattered tracts and islets of a slightly fibrillated, in places almost homogeneous, colloid looking tissue. These tracts enclose vessels which can be distinguished, small in size, and with thickened walls, in the centre of each. (× 150.)

[1] Cf. Perrin and Poncet's "Atlas".—Atrophy of the Optic Nerve.

looking tissue immediately adjacent to the wall of the vessel. It would probably be unjustifiable to assume that this represents the commencement of the process of change. Histology has not hitherto afforded much information as to the initial lesion in these cases. It is on the symptoms that the theory of a primary nerve degeneration is based.

In cases of primary atrophy of the nerve the retina is degenerated only in its inner layers—nerve-fibre and ganglion-cell layer, as Virchow first showed.[1] The other retinal elements may persist in a perfectly normal condition even for many years. Perrin and Poncet could find no change, except in the two inner layers, in a case of ataxy in which sight had been lost for thirty years.

The degeneration from damage to the trunk of the nerve ascends to the chiasma, and descends to the eye. It is long in passing the chiasma, and, even with complete atrophy of one optic nerve, the optic tracts are only slightly reduced in size, that on the side opposite the affected nerve being rather smaller than the other, without naked-eye evidence of degeneration; and I have found that the microscopic changes are nearly equally distributed through the two.[2] When both optic nerves are degenerated the optic tracts may present the same condition, traceable (as Türck pointed out) as far as, and involving, the external corpora geniculata.

Consecutive or Post-papillitic Atrophy. — The microscope shows the substance of the disc to be occupied by nucleated connective-tissue fibres, among which, commonly, few or no traces of nerve fibres are to be discerned. Often, however, the nuclei, by their grouping, indicate the position of the intervals between the fasciculi of former nerve fibres. The retinal layers are displaced outwards (Fig. 21), an important sign of the preceding swelling, and both they and the commencement of the choroid may present some disturbance. The atrophy of the rest of the retina is confined to the inner layer, especially affecting the layer of nerve fibres.

[1] Virchow's "Archiv," vol. x. 1856.

[2] "Centralblatt f. die med. Wissensch.," 1878, No. 31.

Symptoms.—The symptom of atrophy is affection of sight proportioned to the damage to the nerve fibres. The patient becomes conscious of a cloud over objects, which increases; of difficulty in seeing certain minute objects, such as small print: and sometimes of a dark area in some part of the field of vision. Examination shows a change in sight in three directions—(1) diminished acuity of vision; (2) alteration in the field of vision; (3) altered perception of colours.

1. *Diminution in the acuity of vision* is invariable when the atrophy is pronounced; it is almost always more considerable in one eye than in the other. In estimating it care must be taken to ascertain and correct any errors of refraction and defects of accommodation. It may vary from a slight degree to complete loss. It is commonly, but not always, proportioned to the degree of change in the optic nerve visible with the ophthalmoscope.

2. *Alteration in the field of vision* may be of several kinds. It is almost as constant as the diminution in the acuity of vision. The form is commonly a limitation at the margin of the field, progressing concentrically until only a small central area is left, such as is shown in Fig. 52. Such a limitation

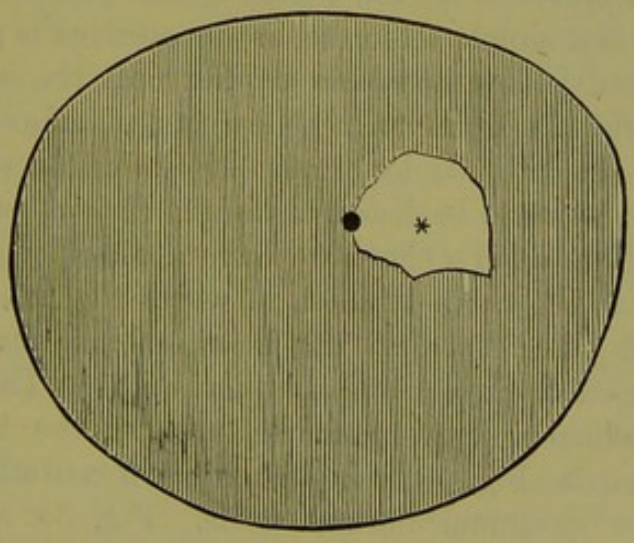


Fig. 52.—Concentric Limitation of Left Field of Vision in a Case of Atrophy of the Optic Nerve.

The outer boundary of the figure is the limit of the normal field. The inner white area is the area of the restricted field.

may progress much more on one side of the field than on the other, or it may progress much more in one part of the field than in another, so as to cause a sector-like defect. Occasionally the diminution is limited to one-half of the field, vertical or lateral. Lastly, in some cases, the first loss is a central one, in the middle of the field, a "central scotoma," as it has been termed. There is often in these cases dimness of the peripheral vision, without concentric narrowing of the field.

3. *Colour-Blindness.*—In many cases the perception of colours is perverted. There are two methods of testing colour-vision. If the patient possesses sufficient intelligence, he may be asked to identify certain colours. If the patient is unintelligent, the "confusion method" must be adopted, by which the colours which are seen alike are ascertained. The former method, however, sometimes gives the more valuable information.

Modern physiological speculation suggests that there are four fundamental colours, related in complementary pairs, red and green, yellow and blue. The area of the field of vision in which these colours are seen varies for each. If coloured objects are moved from the centre of the field to the periphery, the first simple colour to be unperceived is green, the next red, and yellow and blue are lost near the edge of the field for white. Commonly yellow is lost before blue, but sometimes the latter is lost first. If the distance at which each colour ceases to be distinguished in various parts of the field is marked upon a chart, we have a series of concentric lines such as shown in Fig. 53, in which the most internal is the field for green, and the most external the field for yellow, the outer circle being that for white. The amount of light influences very much the area of the fields, and those shown in Fig. 53 were taken upon a dull day, and present the minimum normal fields. Fig. 54 shows the respective fields of larger size, and the blue field the most extensive. Compound colours are lost sooner than their constituents, and the inner circle in Fig. 54 represents the field for violet, which is even smaller than that for green.

Commonly, in atrophy of the nerve, the first defect is for green and red, and blue and yellow are lost subsequently. The order of affection is commonly that in which the fields are arranged on the retina. The simple colour first lost in passing from the centre to the periphery of the retina is that first lost in atrophy, green; and the last to be lost is blue or yellow.[1] Thus a girl, lately under observation, suffering from disseminated sclerosis and commencing grey atrophy, recognized, with the affected eye, every colour except green, which she called red or brown. In another case there was entire loss of perception for green only. Occasionally red appears to be lost first. A patient with ataxy and advanced

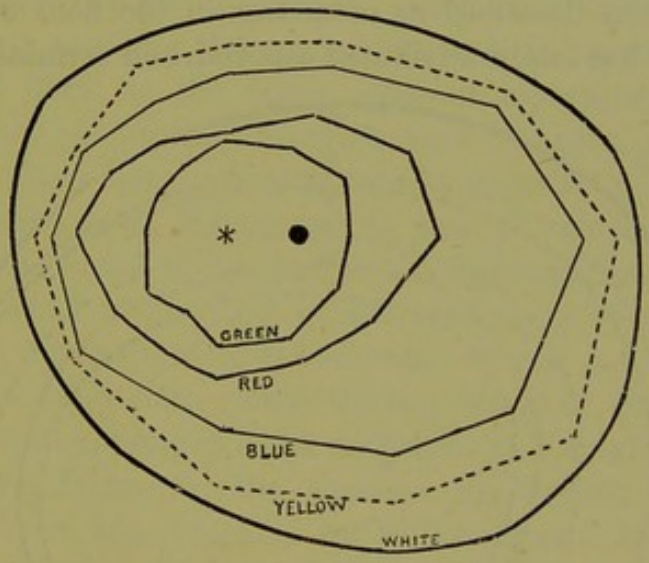


FIG. 53.—A REPETITION OF FIG. 39. DIAGRAM SHOWING THE FIELDS OF COLOUR-VISION IN A NORMAL EMMETROPIC EYE ON A DULL DAY.

The fields are each rather smaller than on a bright day. The asterisk indicates the fixing point, the black dot the position of the blind spot. (Usually the blue field is larger than the yellow.)

[1] It is doubtful whether this is true of violet, which is a compound colour. In some cases (it is said in hysterical amblyopia—Charcot) violet is first lost. Sometimes, however, it persists to the last. Abadie suggests, on the theory that the same fibres conduct all colour impressions, that the first degenerative change in the fibre interferes with its power of conducting the special impression excited by green rays, and the further changes abolish its power of conducting the impressions excited by other rays, in the order above given. ("Ann. d'Oculistique," 1878.)

atrophy (under the care of Dr. Buzzard) stated that the first loss of the sense of colour of which he was conscious, was that he could see no colour in a scarlet geranium. Red gravel looked grey to him. Soon afterwards the grass also looked grey, and he could not, at a little distance, distinguish it from the gravel. When examined, violet alone was seen as a colour, he said it looked blue. A medium blue was seen as white. Cases have also been met with by Uhthoff, Leber, and Treitel in the stage in which perception of red was lost and of green was preserved. The loss of perception of colour is often rather a colour amblyopia than blindness, large pieces of colour may be seen when small spots are not. The fields for colour-vision may present alterations similar to those already described as occurring in the field for white.

Abadie[1] has lately attributed especial, and certainly undue,

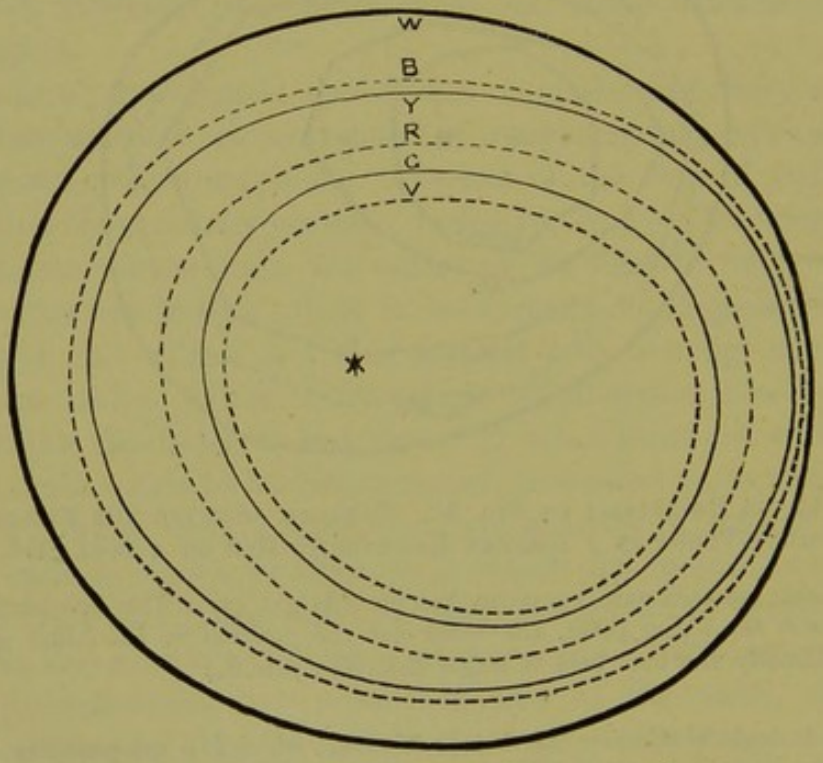


FIG. 54.—FIELDS OF VISION FOR DIFFERENT COLOURS.
(*After Snellen and Landolt.*)

W, white; B, blue; Y, yellow; R, red; G, green; V, violet. These are probably the maximum normal fields for each colour.

[1] "Ann. d'Oculistique," 1878, and Lebris, "Thèse sur les Differentes Formes de l'Atrophie de la Nerf Optique." Paris, 1878.

importance to the loss of colour-vision as a supposed distinction of the parenchymatous from the interstitial forms. It is probably of little significance as regards these forms. The most characteristic loss usually attends interstitial processes.

Relation of Symptoms to Form of Atrophy.—It was suggested by Leber that the central fibres of the optic nerve, on their emergence, probably occupy the most superficial of the nerve-fibre layers of the retina, and have the longest course, while the fibres in the circumference of the nerve lying deepest in the retina end soonest. On this theory a concentric limitation of the field was ascribed to an affection of the axial fibres of the nerve, the central scotoma to that of the circumferential fibres. Förster, however, reversed this theory, ascribing the central scotoma to an affection of the axial fibres of the nerve. Recent investigations have conclusively proved that Förster's view is nearer the truth. Two cases have been published, one by Samelsohn,[1] the other by Nettleship and Edmunds,[2] in each of which a central scotoma was found to be due to the degeneration of a tract of fibres, which at the back of the orbit occupied the axis of the nerve, but in front of the entrance of the central artery lay on the outer side. Thus the hypothesis of Förster that a central scotoma might be an indication of "axial neuritis"[3] is verified. Moreover, the converse verification has been afforded by a case recorded by Wilbrand and Biswanger,[4] who found that a peripheral defect in the field of vision was due to an affection of the circumferential portion of the optic nerve.

Concentric limitation of the field is very common in all forms of atrophy. In the spinal and simple progressive forms it most frequently begins on the outer side, but may commence on the inner side above or below. The acuity of vision may fail at the same time, or may remain normal until

[1] "Centralbl. f. med. Wissensch.," 1880, p. 418.

[2] "Trans. Ophthalmological Society," vol. i. 1881, p. 124.

[3] See Wilbrand: "Klin. Monatsbl. f. Augenheilk.," Dec. 1878

[4] "Centralblatt f. med. Wissensch.," 1879, p. 923, from the "Breslauer Artzl. Zeitschrift," 1879.

the field is reduced to a very small area. When acuity is preserved, if the limitation is regular and sharp, central colour-vision may be normal, but the fields are reduced in area, preserving their normal relation to the field for white. When the limitation, although sharply defined, is irregular, colour-vision is usually much impaired (Nettleship[1]). If, with considerable concentric narrowing, acuity of vision has failed greatly, colour-vision is usually much impaired or lost.

A loss of one-half of the field of vision (apart from cerebral hemianopia) is met with chiefly in secondary atrophy, especially when the cause is pressure on the chiasma, the temporal halves of the fields being then usually lost (see p. 72). But a loss of one-half of the field is met with in rare cases of primary atrophy. Thus in a case of grey atrophy associated with locomotor ataxy, the patient averred that he rapidly lost vision outwards in each eye. When he came under observation there was entire loss of the right field and loss of the temporal half of the left field, the loss including the fixing point (Figs. 55 and 56). Precisely the same affection of sight was present in a case of tabetic atrophy described by Treitel.[2]

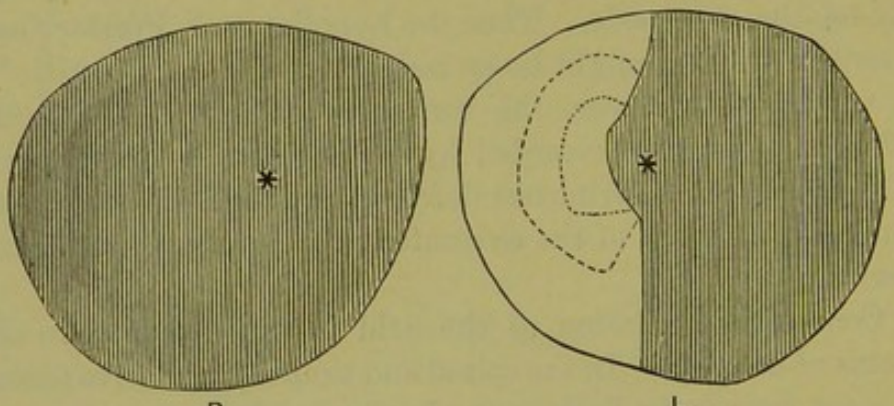


Fig. 55. Fig. 56.

Fields of Vision in a Case of Locomotor Ataxy with Grey Atrophy.

The shading indicates loss. The outer dotted line indicates the field for blue, the inner that for yellow.

1 "British Med. Journal," 1880, ii. 779.

2 "Arch. f. Ophth." vol. xxv. 1879, p. 61.

Sector-like defects in the field are met with in secondary atrophy, especially in cases of injury to the trunk of the nerve at the posterior part of the orbit. They also occasionally occur in simple progressive atrophy and in spinal atrophy. A well-marked instance of this condition in spinal atrophy is shown in Figs. 57 and 58. The patient was in the earliest stage of locomotor ataxy. The optic discs were grey and the vessels small; vision was R. $\frac{1}{12}$, L. $\frac{1}{\infty}$.

Sector-like defects in primary atrophy may be, as in this case, symmetrical, but they are sometimes unilateral, as in a tabetic atrophy recorded by Uhthoff,[1] where in one eye there was a defect of the upper and inner quadrant, and in two cases described by Treitel there was a defect in the inner and lower part in the right eye, as in the case figured.

Central scotomata are usually transversely oval, extending from the blind point to the fixing point, sometimes involving both, sometimes one only. It is uncertain at which point they usually commence. The periphery of the field is usually normal, but it may probably sometimes be restricted. There is always a loss of colour-vision, and this may be greater and occur earlier than that for white. Red and green are first, and may be only lost. Central scotomata for red are

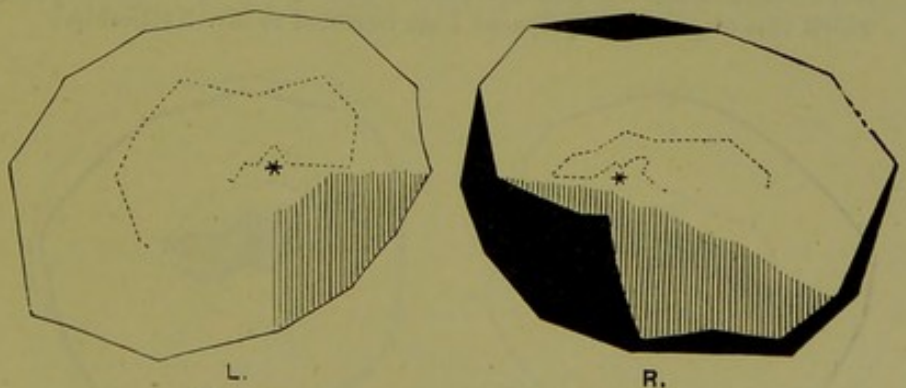


Fig. 57. Fig. 58.

Sector-like Defect in Fields of Vision in a Case of Spinal Atrophy.

The shading represents amblyopia, the black loss. The dotted line shows the boundary of the field for red. Where it is absent the field ceased so gradually that its limit could not be ascertained.

[1] "Arch. f. Ophth.," vol. xxvi. 1880, pt. 1, p. 277.

shown in Figs. 59, 60, and 61. They are met with in cases of axial neuritis and degeneration, and especially in cases of amblyopia from tobacco. That the latter depends on the same

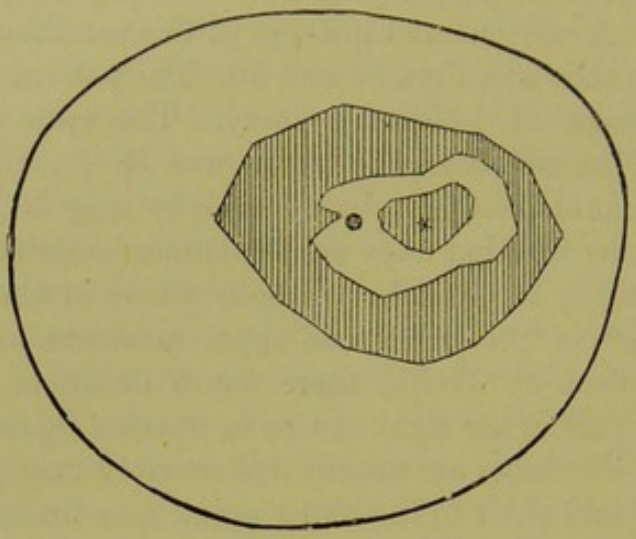


FIG. 59—DIAGRAM OF THE LEFT FIELD OF VISION FOR RED IN A CASE OF TOBACCO AMBLYOPIA.

The outer line is the boundary of the normal field for white. The boundary of the outer shaded area is the minimum normal field for red. Red could, however, be seen only in the inner white area, and it could not be seen in the central shaded area around the fixing point (*). The black dot indicates the position of the blind spot. (For the chart from which this diagram was prepared I am indebted to Mr. Nettleship.)

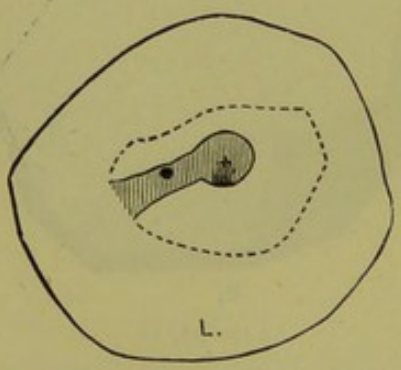


FIG. 60.

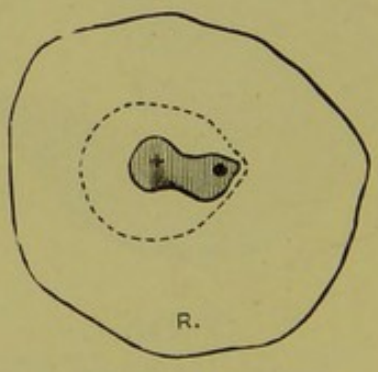


FIG. 61.

CENTRAL SCOTOMATA FOR RED, EMBRACING FIXATION POINT AND BLIND SPOT.

No loss for white but considerable amblyopia (16 Jäger). The patient had smoked half-an-ounce of shag daily. The dotted line represents the peripheral boundary of the field for red. (Nettleship.)

pathological condition (axial neuritis) is probable, both from the character of the affection of vision and from the fact that signs of congestion or slight neuritis are often observed at the papilla. A central scotoma is occasionally met with in consecutive (papillitic) atrophy. It may occur also in the atrophy which succeeds loss of blood, and is probably produced by neuritis. Central loss is occasionally met with in simple progressive atrophy, but in spinal atrophies it is extremely rare—has been said, indeed, never to occur. In a case under my care, however, there was central scotoma (Figs. 62 and 63), associated with the symptoms of lateral and posterior sclerosis of the cord, and some cerebral degeneration; the existence of slight papillitis makes it probable that axial neuritis existed, and, since the patient smoked a little, the influence of tobacco cannot be entirely excluded.

Peripheral areas of vision, with general loss, are met with only in cases of orbital inflammation or in consecutive (papillitic) atrophy. The changes in the latter are often very irregular. There may be general concentric limitation of the field, or, less commonly, a central loss, rarely sharply defined. Failure of colour-vision is very frequent, but is often less regular in order than in primary atrophy (see "Neuritis," p. 71). The colour fields may present very

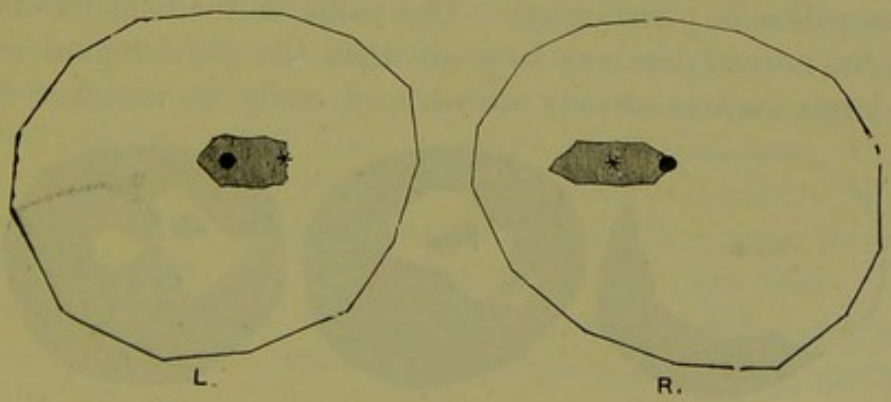


Fig. 62. Fig. 63.

Central Scotomata in a Case of Degeneration of the Spinal Cord.

There was a rather larger central loss for red and green, but the peripheral amblyopia for these colours was also considerable.

irregular defects, as in Figs. 64 to 66, representing the fields for white, red, and green in a case of post-neuritic atrophy. That for yellow and blue was normal, except for a limitation below, and to the inner side, corresponding to the field for white. They were taken nine months after the subsidence of neuritis, when acuity had improved to $\frac{1}{14}$. Uhthoff once met with a central scotoma for blue only.

As a rule there is more or less correspondence between the pallor of the disc and the failure of sight. When it is considered, however, that the tint of the disc depends on its blood-vessels, and the amount of vision on the integrity of the nerve fibres which merely pass through the disc, and have a long course on each side of it, it is not surprising to find that the correspondence between the tint and vision is not always close. A very remarkable case has been recorded by Krenchel,[1] in which the optic discs of a boy became "as white as porcelain," although vision was normal. After some time, however, sight failed with great rapidity.

Diagnosis.—The diagnosis of simple atrophy of the optic nerve rests especially on the change of colour, and the chief difficulty in the diagnosis is due to the degree of pallor sometimes seen as a physiological condition. The existence of amblyopia, otherwise unexplained, is strong evidence that the pallor is pathological. The pallor of the temporal half of the normal disc may be great when the physiological cup is large, and, as already stated, may easily be mistaken for

Fig. 64. Fig. 65. Fig. 66.

Fields of Vision for White, Red and Green in a Case of Papillitic Atrophy.

[1] "Hospitals Tidende," 1878, quoted in Virchow's Jahresbericht," 1878, vol. ii. p. 474.

atrophy. It is certain that many normal cases have been described as "atrophy of the temporal half of the disc." It is doubtful whether an atrophy is ever, except in toxic cases, confined to the temporal half of the disc, in which, ordinarily, the nerve fibres are very few. Although it is true that a slight degree of atrophy may produce the most distinct changes in this half of the disc, yet some pallor is always to be recognized, in such cases, in the nasal as well as in the temporal half. The diagnosis of the congestive variety of atrophy presents greater difficulties, but rests on the uniform distribution of the redness, its soft, velvety surface, the slight blurring of the edge of the disc, in combination with defective vision.

Beginners sometimes mistake the white crescent of "posterior staphyloma" for part of the disc, and thus think the outer part of the disc, the colour of which is of such special significance, is white. Occasionally, especially in myopic eyes, the choroid presents a zone of atrophy, soft edged, around the entire circumference of the disc, which then has an unusual and puzzling appearance. In both these cases, however, attention to the fact that the pale zone encloses a well-coloured disc will prevent mistake as to its real nature.

The excavation which accompanies the pallor is of secondary diagnostic importance, and it is not often that a difficulty in distinguishing atrophy from other forms of excavation arises. It may, however, occur. A large physiological cup may be bounded by a narrow rim of deeply-coloured disc, the boundary of which from the choroid may not be apparent on a cursory inspection by the indirect method of examination, and the large, deep, sometimes grey, cup may be mistaken for the disc. A careful inspection of the edge will prevent doubt, and the examination by the direct method at once shows the source of the error.

The excavation of atrophy commences at the sclerotic ring, and this is a character also of another form of excavation, namely, that of glaucoma. But the depth of the glaucomatous cup, its vertical sides, and the course of the vessels

over the edge, and their subsequent disappearance, are diagnostic, especially since the pain which is so common (though not invariable) in glaucoma is never present in simple atrophy.

Prognosis.—The prognosis of atrophy of the optic nerve, on whatever cause it depends, is always unfavourable in proportion to the actual destruction of fibres which has taken place, and to the extent to which the causes influencing the disease are beyond control. Simple primary atrophy is usually due to a tendency to degeneration beyond all influence, and the prognosis is, in this form, the least favourable. This is especially the case when the atrophy is associated with symptoms of degeneration elsewhere in the nervous system. Secondary degeneration is often the consequence of the operation of causes which may pass away, and the prognosis is less uniformly grave than in primary degeneration. It must, however, always be somewhat uncertain, since it is often very difficult to form an accurate opinion of the nature of the process causing the damage to the nerve, on which the secondary degeneration depends. In the congestive form the prognosis is perhaps rather better than in the other forms. In the atrophy which is consecutive to intra-ocular neuritis, we are able to form a more accurate estimate of the course of the affection by the fact that, as long as the new tissue of the disc goes on contracting, the damage to the nerve fibres increases, and the sight will go on failing. If sight is lost from such contraction some time before it reaches its maximum, the prognosis is very grave. If, however, the loss of sight is incomplete, or only becomes complete when the subsidence is nearly over, some subsequent slow improvement may be hoped for, and this may, in less severe cases, be very great. In a case which I have published elsewhere,[1] for example, probably of tumour in the middle lobe of the cerebellum, there was at first double optic neuritis, with great swelling. On the subsidence of the optic neuritis, six months later, it had diminished in both eyes to

[1] "Trans. Ophth. Soc.," i. 117.

$\frac{1}{15}$. After this the disc atrophied, but at the same time vision improved, until, fifteen months after the first observation, it had risen to $\frac{2}{3}$, and the pupils, which had shown formerly no reaction to light, again acted mormally. Under all circumstances, it is unhappily true that a disc which has lost all its normal tint never regains its vascularity, and useful vision is scarcely ever recovered.

Some prognostic indications may also be drawn from the form of the affection of sight. The gravest, that which indicates not merely damage, but destruction of nerve fibres, is considerable contraction in the field of vision. In proportion as this is extensive the prognosis is grave. Lessened acuity of vision is of less serious prognostic significance. The change in colour-vision is least grave when this depends on a toxic cause, or on neuritis; but is most grave when it is due to a primary degeneration, and occurs early. Central scotomata rarely go on to complete atrophy.

Although the chance of restoration of useful vision in pronounced atrophy is small, in some cases the progress of the disease may be arrested, for a time or permanently, and even improvement obtained, occasionally considerable in degree.

Treatment.—The treatment of optic atrophy is essentially that of the general condition on which it depends—toxic influences; excesses, sexual, physical, mental; cerebral and spinal disease, the "neuropathic constitution," &c. The treatment of many forms of atrophy which are due to an isolated ocular condition is beyond the scope of the present work. Cerebral processes may be to some extent influenced by treatment. Although it is not certain that there is such a thing as an actual syphilitic atrophy, yet atrophy does often result from syphilitic intra-cranial disease, and may greatly improve with the removal, by appropriate treatment, of its cause. Scrofulous brain disease, again, may often be beneficially influenced, and its effects greatly lessened. In other cases counter-irritation, local depletion, purgation, and the like, effect good.

In cases of primary atrophy, which are the result of a

neuropathic tendency, the treatment has to be directed to the general health, and nervine tonics are the chief agents to be employed. Nitrate of silver has been found useful in some cases: in others phosphorus, in others strychnia. The hypodermic injection of strychnia, so useful in amblyopia without ophthalmoscopic signs of atrophy, is of little service where these are present. Quinine and iron are in some cases very useful.

When perception of light is not entirely lost, the retina may be readily stimulated by an interrupted voltaic current, so as to give rise to a sensation of light, and this has suggested repeated stimulation of this character as a means of treating optic nerve atrophy. Some improvement, following treatment with the continuous current, has been observed by Pye-Smith[1] and Gunn[2] in a few of their cases; the experience of others has also been generally unfavourable. I have tried it in many cases, but without results which could reasonably be ascribed to the treatment.

THE RETINA.

Apart from the vessels and the optic disc, the changes in the retina which are of medical importance, are those which are special to certain general diseases, such as syphilis, albuminuria, leucocythæmia, pernicious anæmia, and the like. They will be described in detail in Part II., in the sections on the ophthalmoscopic changes in the several diseases. The only common feature which these morbid states possess, is the development in the retina of hæmorrhages and white spots and patches. The hæmorrhages, their characters and significance, have been already described (p. 25). It may be convenient briefly to describe the forms of white patches which the retina may present under pathological conditions.

A diffuse, slight opacity of the retina may be due to the derangement of its normal structure, resulting from the effusion of serum among the structures which compose it. Such diffuse opacity occurs in embolism, neuritis, and

[1] "British Med. Journal," May 18, 1872.
[2] "Ophth. Hosp. Rep.," vol. x. pt. 2, June, 1881, p. 161.

albuminuric retinitis, but in all, and especially in the latter, it is usually associated with structural changes. Circumscribed opaque white spots are due to change other than that of simple œdema, and commonly of four varieties: (1) Fibrinous exudations which undergo coagulation; (2) the accumulation of corpuscles, similar in appearance to those of the nuclear layer, and also to the white corpuscles of the blood, so that it is doubtful from which source they are derived; (3) fatty degeneration of the retinal elements, perhaps also in part of fibrine from the serum effused in simple œdema, and of the remains of blood clot; (4) a fibroid change, a process of "sclerosis" of the retinal elements is described as an occasional cause of a white spot, but is more frequently confined to the perivascular tissues and vessel-wall.

These conditions are frequently combined. The fatty degeneration may exist alone, as the sole cause of a white spot. Corpuscular accumulation usually involves a good deal of fatty degeneration in the cells and in the disturbed retinal elements. Sclerosis of the retinal structures is also in most cases associated with fatty degeneration.

It is often impossible to say, from the ophthalmoscopic appearance, on what change the white spot depends. Minute granular-looking spots, brilliantly white, are commonly due to fatty degeneration of retinal structures or of leucocytes, &c. Larger white spots, if soft edged, are commonly effused fibrine or accumulations of leucocytes, especially if situated beneath the nerve-fibre layer. Fatty degeneration of the retinal structures is, however, commonly associated. White spots in the superficial layer of the retina, most common in Bright's disease, are due usually to degeneration of the nerve fibres.

Growths in the retina sometimes occur in cases in which there are other growths elsewhere. The disc shown in Pl. III. 4 was from a boy who had cerebral tubercles, and whose other eye was the seat of a tubercular growth behind the retina.

The occurrence of miliary tubercles of the retina has been suspected by many observers. White spots are sometimes seen adjacent to the disc in cases of tubercular meningitis, and such a spot in one case I found to be made up of

lymphoid cells like those of the nuclear layers in which it was situated. Bouchut has seen white spots at a distance from the disc, near the vessels. Microscopically, he always found them to contain only products of fatty degeneration. He suspected them to be caseous tubercles, but there was no direct evidence that this was their nature (see Part II.).

Since the white spots in the retina which have been described, are present in many forms of retinal disease which occur secondarily to, and are significant of, general diseases, it is of great importance to distinguish them from other appearances which have a different significance.

First, it is necessary to distinguish whether the white spot is in the retina or in the choroid. Most choroidal white spots are due to atrophy of the choroid, and their distinction is easy. The atrophy of the choroidal pigment permits the white sclerotic to shine through; some choroidal vessels may have escaped destruction and course across the white patch; its edge is always more or less irregular, and usually much pigmented; or the choroid may exhibit adjacent slighter disturbance. It is easy to recognize by the "parallactic test" (also in the direct method by attention to the time required for the necessary change of accommodation), that the exposed sclerotic is some distance behind any retinal vessels which pass in front of it. Occasionally, however, a white spot in the choroid is due to a recent formation, an inflammatory "exudation," or a growth such as tubercle. This is prominent, and may be difficult to distinguish from a white spot due to change in the nuclear layers of the retina. In some cases a little pigmentary disturbance in the neighbourhood may be seen; in others the white surface is distinctly so far behind the retinal vessels as to be obviously at the choroidal level. If sufficiently prominent to disturb the course of the retinal vessels, the prominence may be recognized and seen to be considerable in degree. In other cases, the conditions of origin of the spot may assist the diagnosis. It must be remembered that large choroidal exudations may cause opacity of the overlying retina.

White spots due to the persistence of the white substance

of the retinal nerve fibres, or to connective tissue at the back of the vitreous, may be mistaken for new formations in the retina. They have been already spoken of. Pigmentary deposits in the retina may be left after extravasation of blood, but such are always small. More extensive pigmentation is commonly the result of the accumulation in the retina of its disturbed pigment, and is a consequence of choroido-retinitis, or it is a result of the so-called retinitis pigmentosa.

Retinitis pigmentosa appears to have some obscure connection with morbid states of the nervous system. It occurs, as Liebreich first pointed out, very frequently in the offspring of marriages of consanguinity. It has been thought to be connected with inherited syphilis, but the evidence on the subject scarcely supports the theory. It often occurs, however, in families in which there is a history of nervous disease. This is well illustrated by three out of four cases of the disease narrated by Mr. Nettleship.[1] Of the first patient, two cousins were epileptic and two insane. Of the second, the grandfather and great aunt were insane, and an aunt half imbecile, and a brother paraplegic. Of the third patient, the mother was epileptic, and probably also suffered from retinitis pigmentosa.

THE CHOROID.

Choroidal changes, like those of the retina, are for the most part the result of special diseases, and their characters will be described in greater detail in Part II. Hæmorrhages are rarely seen, although their consequences are sometimes met with. The common changes consist in white spots and the disturbance of the choroidal pigment, which so constantly results from any changes in its structure. The white spots are either new formations or patches of atrophy. The distinctions between them have just been alluded to in describing the diagnosis from retinal changes. White spots, not atrophic, are the result of inflammation, or growths—tubercle or lymphadenoma. The latter are extremely rare, and only occur when the general lymphatic disease is well marked. Tubercles are isolated and small—rarely large. Pigment

[1] "Ophth. Hosp. Rep." ix. 170.

may be seen adjacent to the older formations. The evidence of the general disease is almost always so prominent as to prevent the possibility of error. In acute choroiditis the white patches are large and numerous: the signs of the dyscrasiæ associated with growths are absent, and there is often a well-marked history of syphilis. The results of previous choroiditis are very conspicuous atrophic and pigmentary changes, often associated with pigmentary deposits in the retina. It must be remembered that this pigment frequently occupies only or chiefly the peripheral portions of the choroid, and an examination confined to the neighbourhood of the optic disc may be insufficient to discover it. The changes are very important, on account of the frequency with which the inflammation causing them is the result of syphilis. They are also interesting to the physician as associated, in some other cases, with evidence of a family tendency to nervous disease.[1] It is possible that inherited syphilis may be the link between these morbid states.

Choroidal exudations (local) sometimes occur about puberty, resembling choroidal tubercles, and it has been suggested that these are really foci of scrofulous or tuberculous inflammation.

Chronic choroidal degenerations sometimes occur as a senile change, possibly in consequence of general arterial degeneration.[2] Circumscribed changes may result from hæmorrhage. Amyloid degeneration of the choroidal arteries was found by Knapp in a case in which hæmorrhage occurred.

Embolism of choroidal vessels was believed by Knapp to be the cause of morbid appearances in two cases of heart disease observed by him.[3] In each there was sudden affection of sight, at first general and then central, accompanied by achromatopsy. Corresponding to the scotoma, there was a localized retinal opacity with hyperæmia. The opacity, ascribed to effusion, extended to the optic disc. Sight, and the appearance of the fundus, ultimately became normal.

[1] Instances of this have been related by Mr. Nettleship. ("Ophth. Hosp. Rep.," ix. 178.)

[2] Hutchinson and Tay: "Ophth. Hosp. Rep.," vol. viii. Poncet: "Ann. d'Oculist.," 1875.

[3] "Arch. f. Ophth.," Bd. xiv.

PART II.

OPHTHALMOSCOPIC CHANGES IN SPECIAL DISEASES.

DISEASES AND INJURIES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

In diseases of the brain, two forms of ophthalmoscopic change may be met with:—Firstly, those which are a consequence of the general condition by which the cerebral disease is produced—*associated changes;* and, secondly, those which are the consequence of the cerebral disease—*consecutive changes.*

Anæmia and Hyperæmia of the Brain.

It has been supposed that the state of the circulation in the eye and brain correspond, and that the anæmia and hyperæmia of the brain are revealed by similar conditions in the fundus oculi, and especially in the vessels of the retina and optic nerve; the vascularity of the choroid being too great to permit of the recognition of any change in its circulation. But, as already stated (p. 19), this conclusion, if true at all, is true only within narrow limits. Local influences, chiefly perhaps the intra-ocular tension, so influence these vessels, that they undergo little alteration when changes occur in the condition of the vessels of the brain. The eyeball participates in variations in the blood-supply to the whole head, but it does not share simple

vascular states of the brain (in which the rest of the head does not participate) to a degree that can render it an index to the existence of those states. This statement applies especially to the retinal vessels: it is in these that alterations can be most readily perceived. It applies also to the optic nerve; but in this, alterations are more readily produced by encephalic changes, although to only a slight extent and not, perhaps, in a direct manner.

Cerebral Hyperæmia.—There is no sufficient evidence to show that the vascularity of the disc or retina participates in any transient cause of cerebral congestion, unless the whole head suffers. But in some cases of long-continued vascular disturbance, and in morbid states which are ascribed, with some probability, to cerebral congestion, ophthalmoscopic changes are sometimes to be seen—a transient increase of colour, sometimes with slight blurring of the edge. But in most of these cases there is evidence of grave functional disturbance of the brain or prolonged hyperæmia. Instances are such congestions as are shown in Pl. I. 1 and 2, and the bright injection of the discs described by Macnamara as occurring during the headache produced by exposure to the tropical sun, increasing to papillitis when actual meningitis is developed.

This conclusion—the absence of any marked vascular alteration in the eye in changes in the cerebral circulation—is at variance with early statements and *a priori* theories; but it is abundantly supported by skilled observers.[1]

[1] See, for instance, the statements of Manz, Schmidt-Rimpler, and others, at the discussion at Heidelberg, reported in the "Ann. d'Oculistique," vol. lxxiv. 1875, p. 262, *et seq.*

It must be remembered that "congestion of the brain" as a name is exceedingly convenient, especially to those who are called "ready diagnosicians," but for whom "unscrupulous namers" would be a more exact designation. Apart from these, however, the condition is invoked with a readiness that cannot but excite surprise in those who know how different is the significance of the symptoms it is considered to cause. Further, the evidence that may suffice for "practical diagnosis" is often wholly inadequate for scientific reasoning. Very red discs, simply suggestive of cerebral congestion alone, prove nothing. To be of significance the redness must lessen in an unequivocal degree as the symptoms go.

Lastly, it is *probable* that when cerebral hyperæmia is due to blood states, the cause may also influence the optic disc and induce congestion. But this has not yet been proved.

Anæmia of the Brain is rare as a primary vascular condition, except as part of a general cephalic anæmia. It is possible that in the same stage of an epileptic fit in which there is pallor of the face, there may also be pallor of the disc; but no evidence of this fact has at present been obtained, and it is unlikely. Indeed, it is questionable whether any diminution in the tint of the disc has ever been observed to coincide with a diminution in the amount of blood within the brain alone. It is not probable that there is such a diminution at the onset of an epileptic fit; the pallor of face usually precedes instead of following the onset.

When the cerebral anæmia is part of a similar state affecting the whole head, the retina certainly participates, although it is not often that an opportunity is obtained of observing this with the ophthalmoscope. But loss of function of the retina affords evidence of its participation; transient loss of sight, probably from this cause, may follow syncopal seizures. In an instance that came under my observation, a lad engaged in a stooping occupation in a hot crowded room, felt faint, and went out into the cool night-air. On re-entering the room he could not see: the room was absolutely dark to him. After sitting still for a few minutes sight slowly returned. It is hardly conceivable that the loss of sight was the result of anæmia of the brain, because the other cerebral functions were scarcely affected, and the loss of sight persisted after he otherwise felt quite well. Probably the retina shared the cephalic anæmia (due to heart-failure), and suffered in function more and longer than the brain.[1]

[1] This fact is one of some significance. It suggests how extremely sensitive the retina is in its function, and therefore must be in its nutrition, to sudden influences.

Inflammation of the Brain.

Acute general inflammation of the brain cannot occur except in association with meningitis. The latter is the predominant lesion, and to it the symptoms are customarily ascribed. Certainly, of ophthalmoscopic changes in acute inflammation of brain without meningitis, we know nothing. Of course such cases of "active hyperæmia" as those described in the last section, as occurring from the effects of insolation, may be regarded as cases of encephalitis. There is no sharp line to be drawn between "active congestion" and "inflammation," but there appear to be no pathological facts to warrant us in regarding the morbid process in these cases as actual inflammation. Local acute inflammation is probably always secondary. But it is probable that any local inflammation of the brain will cause neuritis if it continues for a sufficient time.

There is, however, a class of cases to which the term "chronic encephalitis," or, perhaps, more accurately, "chronic cerebritis," appears fully applicable, and in which there may be very marked ophthalmoscopic changes. These cases present evidence of mental and motor failure, the latter may be local and attended by convulsion. Death may be preceded by coma. Headache is often severe. There are not the tremors or mental peculiarities of general paralysis, the symptoms resembling much more closely those of cerebral tumour. Post-mortem there is no sign of meningitis; the brain may present evidence of degeneration, sometimes of wasting, but no "focal" disease. Such cases may be attended by optic papillitis very similar to that found in cerebral tumour, due most probably to the propagation of an irritative process from the cerebrum along the nerves. A well-marked case of this kind has been described by Hughlings-Jackson.[1] Dr. Sutton's microscopical examination of the convolutions showed

[1] "Ophth. Hosp. Rep.," viii. 445.

only an undue number of the "spherical nuclear bodies," and in places, instead of the normal pyramidal nerve cells, were large numbers of staining nuclei, with unstaining cell-bodies around them. In places these nuclei were aggregated into groups of ten or twenty. The neuroglia was more granular than that of a healthy brain. The optic nerves, examined by myself, presented the characteristics of moderate papillitis, the swollen papillæ being infiltrated with nuclear bodies similar to those seen by Dr. Sutton in the brain. Similar corpuscles were so abundant throughout the optic nerves as to justify the assumption that the neuritis had been "descending" (Figs. 47, 48). A case published by Noyes, in 1873, was probably similar. Double optic neuritis, passing into atrophy, was accompanied by severe pain in the head, and paralysis of various cerebral nerves and unsteady gait. After death, no lesion of the brain was discovered. More recently, a well-marked case of the same kind, also accompanied by optic neuritis, has been recorded by Stephen Mackenzie.[1]

In the rare cases in which hæmorrhage, or softening from vascular occlusion, causes optic neuritis, the effect is doubtless produced through the agency of secondary inflammation.

Cases are sometimes met with in which we have a difficulty in assigning to inflammation or growth the chief share in the morbid process. Such cases may be accompanied by descending neuritis, and simulate closely the symptoms of cerebral tumour. Pl. VI. Fig. 2 shows the optic disc in such a case. In this case, local injury, years before, had caused the production of cheesy degenerating tissue beneath the membranes over certain convolutions, and a more widely spread but irregularly distributed meningitis had led to vascular disease, from the effects of which the patient died. The optic nerves were infiltrated with leucocytes, and "miliary abscesses" were found in the optic tracts (Figs. 23 and 33).

[1] "Brain," vol. ii. p. 257.

Cerebral Hæmorrhage.

Associated Changes.—The common form of cerebral hæmorrhage is due to the rupture of "miliary aneurisms"; that is, minute arteries suffer in the nutrition of their wall, which yields before the blood-pressure, and the dilatations thus produced are called aneurisms. The conditions which give rise to these aneurisms seldom influence the arteries of the retina, but the capillaries suffer in a similar manner very frequently, and thus small extravasations occur. In Fig. 3, p. 17, are shown capillary aneurisms from a case in which cerebral and retinal hæmorrhages coexisted. These associates, retinal and cerebral aneurisms, occur, however, almost exclusively in kidney disease. Aneurisms on small arteries, the true "miliary aneurisms," are rare. Probably this is due to the uniform support afforded to the arteries of the eye. Aneurisms are also depicted in Pl. XII. from a case in which all the conditions for the production of cerebral hæmorrhage were present in extreme degree. It is taken from a woman aged thirty-six, who had advanced kidney disease with great cardiac hypertrophy, and very high arterial tension. On ophthalmoscopic examination there was obvious change in the coats of all the branches of the retinal artery—chiefly thickening of the outer coat. There were several large hæmorrhages, and in a few places aneurismal dilatation of the vessel.

Retinal Hæmorrhages, however, are present in a considerable number of cases of cerebral hæmorrhage, and furnish an indication of considerable value. Their most frequent cause is that which is the most frequent cause of cerebral hæmorrhage, Bright's disease, especially the granular kidney. They may exist, as in Pl. IX. 1, without any other retinal change, or may form part of the special retinitis (Pl. X. 1 and XII. 1). In either case they indicate the existence of the conditions which favour vascular degeneration and rupture. In the retina shown in Pl. X. 1, for instance,

capillary dilatations and other changes were found. The retinal hæmorrhages are often associated with cardiac hypertrophy. They thus may accompany all the most potent causes of cerebral hæmorrhage. It must not be concluded, however, that the presence of albuminuric retinitis proves a cerebral lesion to be hæmorrhagic. The disease of the kidneys is a cause, not only of the minute aneurisms that lead to hæmorrhage, but also of the atheroma of the larger arteries that leads to thrombosis within them. Hence, softening due to the closure of atheromatous arteries is often associated with retinal changes due to kidney disease, and the latter have little weight in the differential diagnosis. They point strongly to one of these two lesions, but leave uninfluenced the indications furnished by the state of the heart and the character of the onset.

In other conditions retinal hæmorrhages have a similar significance. They point to a state in which cerebral hæmorrhage is likely to occur. Their significance is also more decided, since these other causes of retinal hæmorrhage do not produce arterial atheroma. They occur, for example, in pernicious anæmia (Pl. XI. 1) and in leucocythæmia (Pl. XI. 2), and in the latter disease the brain stands second in frequency as the seat of internal hæmorrhage.[1]

But although retinal hæmorrhages point to the existence of conditions such as may lead to cerebral hæmorrhage, and are thus of great importance as indications of the need for care in avoiding the exciting causes of hæmorrhage, their significance as indications of the probability of the occurrence of apoplexy may be overrated. They are not uncommon, as in old and gouty persons, who do not suffer subsequently from cerebral hæmorrhage. Perhaps this is, in part, due to the fact that the conditions in which they arise are such that many other causes of death coexist. Moreover, the existence of the conditions favourable to an event does not necessarily involve a balance of probability in favour of the occurrence of that event.

[1] Retinal hæmorrhages not included. See the writer's article on "Leucocythæmia," "Reynolds' System of Medicine," vol. v.

Among the very rare causes of cerebral hæmorrhage are syphilis and heart disease; the occasional changes in the fundus may thus conceivably be of service in the differential diagnosis, that from heart disease being embolism. The coincidence has not, however, been hitherto observed. One reason for this may be that the hæmorrhage from these causes is usually due to the rupture of an aneurism, and is seldom survived.

Consecutive Changes.—Hæmorrhage into the substance of the brain is not usually attended with any ophthalmoscopic changes. So rarely have any alterations in the fundus been seen, that they may be said almost never to occur during the first few weeks after the onset. Neuritis has, however, been met with in a few instances. But its rarity is so great that the question arises whether, when met with, it has really been due to the cerebral lesion; and the question is certainly justified, because other causes of optic neuritis are seldom absent in cerebral hæmorrhage. Kidney disease and constitutional gout are sufficient to explain the occurrence of optic neuritis when it exists alone, and equally so when it is met in association with a malady that has not yet been proved to be, alone, an adequate cause. But here, as in so many other conditions, the insufficiency of a cause acting alone, does not exclude some influence when it is in association with some other cause. There is evidence that inflammation of the brain will produce optic neuritis, and that any process that excites secondary inflammation may therefore assist in the causation. Hæmorrhage always causes such secondary inflammation, just as does a traumatic lesion of the brain. Indeed cerebral hæmorrhage may be regarded as a traumatic lesion which has no external origin. Although the secondary inflammation seems to be insufficient, alone, to excite optic neuritis, it may determine the occurrence in conjunction with so potent a cause as the blood-state of gout, or that produced by kidney disease. This is probably the explanation of the few cases in which considerable optic neuritis has been observed in cases of pure hæmorrhage. One such case has been described by Hughlings-Jackson; ten weeks after an attack of cerebral

hæmorrhage, the discs presented the appearances of the later stage of neuritis. The patient died a week subsequently, and the necropsy revealed a large extravasation into the middle cerebral lobe, and a few specks of hæmorrhage into the corpora quadrigemina. Another case is recorded by Bristowe.[1] The hæmorrhage was in the posterior part of the optic thalamus. Robin[2] mentions a case with well-marked neuritis, such as is met with in tumours, in which the autopsy revealed a clot of blood, the size of a walnut, compressing the pons. In this case the neuritis can hardly have been the result of the extravasation. In a case described by Gemuseus,[3] double neuro-retinitis was observed during life, and, after death, numerous hæmorrhages were found in the brain.

In many cases of intense optic neuritis met with in cerebral hæmorrhage, the blood has been extravasated into a soft growth in the brain, to which the neuritis has really been due. In one case, in which the neuritis had been watched during life, a careful observer who made the post-mortem reported a large clot surrounded by secondary softening, but the latter was really a very soft grey glioma into which the hæmorrhage had occurred.

When, however, the hæmorrhage is into the meninges, ocular changes may exist—slight optic neuritis. The hæmorrhage may pass into, and distend, the sheath of the optic nerve, as has been found (in a case of my own) in meningeal hæmorrhage from fracture of the skull, in rupture of an aneurism of the middle cerebral (Mackenzie), in rupture of an intra-cerebral extravasation into the meninges (Michel), and in hæmorrhagic pachymeningitis (Manz). Opacity and blurring of the outline of the disc with slight swelling may be thus produced. Retinal extravasations may co-exist, as in a case figured by Poncet.[4] Early changes in the papilla, in a

[1] "Trans. Ophth. Soc.," vi. 363.

[2] "Des Troubles Oculaires dans les Maladies de l'Encephale," Paris, 1880, p. 284.

[3] "Klin. Monatsbl. f. Augenheilk.," 1880, p. 380.

[4] "Atlas" of Perrin and Poncet.

case of undoubted cerebral hæmorrhage, would thus be evidence that the blood was effused into the meninges. It is said (by Knapp and Liebreich) that a peculiar pigmentation of the outer peripheral part of the disc, within the sclerotic ring, may be an ultimate consequence of such hæmorrhage.

In rare cases optic nerve atrophy has been met with in association with cerebral hæmorrhage. Thus a case is recorded by Vulpian[1] in which blindness supervened on an attack of apoplexy. Death occurred fifteen years later, and the remains of an old hæmorrhage were found in the left corpus striatum. Both optic nerves and optic tracts presented grey atrophy. The connection between the two is probably indirect.

Cerebral Softening.

In softening of the brain, marked ophthalmoscopic changes are rare as the result of the cerebral mischief, although occasionally present, as several reliable cases testify; and I believe that slight changes are more common than in cerebral hæmorrhage. Most of the cases in which alterations have been found have been cases of softening from embolism, not from thrombosis secondary to vascular disease. Changes in the fundus oculi, moreover, sometimes result from the same causes as those which lead to the cerebral mischief.

1. Embolic Softening: (*a*) *Associated Changes.*—Embolism of the trunk or of a branch of the central artery of the retina may occur before or after embolism of a cerebral artery; very rarely at the same time (see p. 34.). When the two occur at the same moment, the demonstration of the nature of a cerebral lesion is brought almost to its most complete form. The only defect in the demonstration is that the plug in the artery cannot actually be seen. An instance of such coincidence is afforded by the case illustrated in Pl. XII. 2. In this, however, the proof was completed

[1] Galezowski: "Journal d'Ophthalmologie," Jan. 1872.

by post-mortem inspection. The plug in the retinal artery is shown in Fig. 4, p. 36.[1]

(*b*) *Consecutive Changes.*—When the artery plugged is the middle cerebral, marked disturbance of the circulation might be expected in the eye which derives its blood-supply from the same trunk. Any signs of such disturbance have, however, hitherto escaped attention, and probably the free anastomoses of the circle of Willis carry off any excess of pressure.

If the condition of the discs is carefully observed from time to time, I believe that a state of congestion may often be observed a few weeks after the onset of embolic softening, especially in those cases in which the cerebral damage is extensive and leads to mental change. Pl. I. Figs. 1 and 2 show such an appearance in a young man with mitral disease and left hemiplegia.[2] Actual neuritis has been observed in a few cases, distinct, moderate in intensity, coming on a few days or weeks after the cerebral lesion, running a subacute course, and slowly subsiding. One of the best marked cases of the kind has been recorded by Broadbent,[3] in a man, aged nineteen, with mitral disease, who was seized with left hemiplegia and impairment of sensation. Nine days after the onset of the hemiplegia the margins of the optic discs were ill-defined; there was swelling, with an unduly vascular "woolly" appearance, the retinal veins were large, dark, and tortuous, the arteries visible, but small. By the eighteenth day the paralysis had improved considerably, but the papillitis persisted, sight being normal. Six weeks after the onset, he was walking about the ward, and the papillitis was subsiding. A fortnight later the outlines of the discs were becoming perceptible,

[1] For fuller details see description of the plate.

[2] The increased redness of the disc, with slightly softened outline to indirect image, developed in both eyes under observation, and was so marked that I thought neuritis was coming on. It became stationary, however; soon lessened in the left eye, and much more slowly in the right. Coincidently with it there was marked and increasing mental failure, persistence of the complete hemiplegia, and rapid development of the ankle-clonus.

[3] "Clin. Trans.," vol. ix. 1876, p. 62

the papillæ being still red and rather prominent. He subsequently had some convulsive attacks and symptoms of ulcerative endocarditis, and died four months after the onset of the hemiplegia. There was softening below the posterior cornu of the right lateral ventricle, extending to the tip of the occipital lobe, and involving the tail of the corpus striatum and the fibres passing from the thalamus to the occipital lobe. The part softened was in the region of the posterior cerebral, but no obstruction of this vessel was found; the calcarine artery could not be traced.

Double neuritis, with slight changes in the contiguous retina, was seen by Stephen Mackenzie[1] in a case of left hemiplegia, no doubt the result of embolism of the right middle cerebral artery. The softening found five weeks after the onset was slight, and the middle cerebral was pervious, although thickened, the probability being that the plug had broken up and had been carried on into some of the terminal branches of the artery. Splenic infarctions were also found. Three days after the onset, the discs (previously normal) were swollen, and three weeks later the swelling persisted, but with a good deal of opacity, the vessels being "buried in exudation." One or two hæmorrhages existed close to the discs. The appearance of the discs, Dr. Mackenzie has informed me, was precisely that often seen in cerebral tumour.

A grey infiltration, incompletely veiling the disc, and extending into the adjacent retina, is figured by Bouchut from a case of hemiplegia in a child of seven years with mitral regurgitation.

Most of the above cases seem to be distinct instances of the association of neuritis and softening. It is important to note that all were cases of softening from embolism, that in most the plug came from valves the seat of actual recent inflammation, and that in some the development of the optic neuritis was accompanied by evidence of wide-spread disturbance of the cerebral functions. There is nothing in the mere process of necrotic softening, the mere breaking up of the nerve-elements into discontinuous particles separated

[1] "Brain," Jan. 1879.

by liquid, which can cause optic neuritis, according to our present knowledge. But the process is never one of simple necrosis of the tissue-elements. Adjacent inflammation always accompanies it just as it does the suppuration of a gangrenous foot. Inflammation in parts of the body shows wide variations in its character, variations which at present are imperfectly understood but are certainly of vast importance. One of the differences in character is the tendency to spread. It is certain that emboli from an inflamed cardiac valve have a special power of exciting inflammation—a power which is ascribed, with much plausibility, to the presence of organisms within them. The inflammation thus excited or conveyed varies in its intensity and in its tendency to spread, just as the inflammation in the heart seems to vary in its "malignancy." It is inevitable, therefore, that in some cases the inflammation that is secondary to the process of necrosis should have its character modified by the influence of the plug; the organisms of it, if organisms are the morbific agents, may readily find their way into the inflamed brain tissue and determine characteristics possibly more extensive than the immediate influence of the presence of the organisms themselves. And it is thus inevitable that the cases should present such differences as we have noticed, and that in some the spread of the irritative process should lead to an optic neuritis, slight or severe, which is absent in other cases.

It may be well, however, again to remark how easily the error may be made of mistaking a soft glioma for a patch of softening.[1]

[1] The following case has been recorded by Drs. Darby and Upham ("Boston Med. and Surg. Journal," vol. lxxii.) as one of softening, in which, however, there was no evidence of embolism. A man aged twenty-six had a hemiplegic attack, followed by fits and double "neuro-retinitis" with hæmorrhages. A necropsy some months later revealed a peculiar softening of the corpus striatum and optic thalamus, grey and white gelatinous soft tissue, to the naked eye very like a glioma, but, on microscopic examination, only the signs of degeneration were visible. It is to be remarked, however, that many parts of these tumours may contain, and even appear to consist only of, products of degeneration. A careful search may be necessary for the very delicate cells of which they consist.

In a case recorded by Leber of supposed neuritis from softening, the fact that the "softening" was a soft glioma was not suspected; the nature of the lesion was only discovered on microscopic examination.

Atrophy of one optic nerve is said to succeed softening, embolic or other, just as it has been observed to succeed hæmorrhage. This result is supposed to be due to the seat of the lesion being such as to damage the nutrition of some part of the brain to which the optic fibres are related. Embolism of the middle meningeal artery, which supplies the dura mater near the optic nerve, is said also to cause atrophy of the latter.

2. Softening from Thrombosis.—(1) *Arterial.*—This may be due to syphilitic or degenerative disease of the vessels, or to blood-changes.

Syphilitic Disease.—In softening from syphilitic disease of vessels, associated ophthalmoscopic changes are common, consecutive changes are very rare. The associated conditions are the various changes which are due to syphilis, and which need not be mentioned here. They come practically under the cognisance and teaching of the ophthalmic surgeon. This is because their active stage affects sight and seldom coincides with disease elsewhere. But the changes in the eye in inherited syphilis come very often under the notice of the physician, and in both the inherited and acquired diseases the relics of the syphilitic disease are of extreme value to the physician. Among the diseases in which their significance is of the greatest importance are those now under consideration.

In cerebral softening such indications are, of course, of the greatest significance in the case of persons who have not reached the period of life at which vascular degeneration is common. In the latter condition, the recognition of constitutional syphilis still leaves us in some doubt, and care must be taken to avoid attaching undue weight to its signs. Causal indications are of significance, in general, in proportion to their isolation. At the same time it must not be forgotten

that syphilitic vascular disease does occur, and not rarely, in the degenerative period. Syphilitic disease and atheroma have been observed post-mortem in the same individual. In doubtful cases, the recognition of the ocular signs of syphilis should always lead to a trial of the special remedies.

Although associated changes are common, consecutive alterations in the eye are very rare in softening from syphilitic disease of vessels. Only one case has yet been recorded in which congestion or inflammation of the optic papilla was apparently due to this cause. The proof can only be given by pathological demonstration of the absence of any other morbid process. The case is one described by Leyden, but it is not quite conclusive, since the inflammation of the papilla may have been a primary lesion.[1] In the few recorded cases in which such changes were observed, syphilitic growths in the brain were associated with the vascular disease, and the ocular change was due to the former, not to the latter. I have met with one case in which a fortnight after the sudden onset of hemiplegia, in a patient who had had constitutional syphilis, there was slight distinct optic neuritis; but the absence of growths could not be excluded, and preceding pain in the head for six months rendered it probable that there was more than arterial disease.

In all cases of this kind the question arises, can the coincident papillitis be an independent effect of the syphilitic poison? This question we cannot at present answer with a positive negation. Syphilis probably can cause a retro-ocular neuritis; it certainly can cause retinitis involving the papilla. Isolated double papillitis may be an effect of many morbid states of the blood, varied in character, with a virus

[1] "Zeitsch. f. Klin. Med.," 1882, Bd. ii. p. 173. The patient, a man aged eighteen, died from limited softening of the inner part of the right crus and adjacent part of the pons, due to syphilitic disease of the extremity of the basilar artery; and seven days after the onset of the acute symptoms there was found "neuro-retinitis with choked disc as in cerebral tumour" (Dr. Hiller), although no other lesion than syphilitic disease of the vessels could be found. The details from an ophthalmoscopic point of view leave much to be desired.

organized or inorganic, and cases are met with possibly presenting a pure syphilitic papillitis. Thus this possibility constitutes at present an unbridged break in the proof that softening from syphilitic disease of the vessels causes papillitis. The difficulty is the greater in proportion as the papillitis is intense. It then exceeds the degree met with even in softening from irritative embolism, and on the other hand resembles that of which the chief causes are tumours and blood-states.

Degenerative Disease: "*Atheroma.*"—Cerebral softening from this cause is rarely associated with any similar morbid state of the retinal arteries, which are below the size in which "endarteritis deformans" is common. Occasionally, thickening of the wall or undue tortuosity of the retinal arteries has been observed. But it is doubtful whether the appearances that have been described as atheroma are really such, or if this state has ever really been met with. The malady is an affection of the inner coat, and such alterations as are depicted in Pl. XII. Fig. 1 are manifestly seated in the outer coat of the vessel or in its sheath. Changes in the retina in the old have been ascribed to atheroma lessening the blood-supply, but such an inference has, of course, no bearing on the question whether atheroma occurs or not.

Atheroma of cerebral vessels is very common in cases of chronic kidney disease, and it is in them that these appearances have been chiefly seen, but this does not prove their nature. Various elements in the retina suffer in renal disease, and hence all forms of albuminuric retinitis may be associated with cerebral softening. They are also associated, in the same manner, with cerebral hæmorrhages, and hence the affection of the retina and even hæmorrhages in it are evidence only of probable disease of the arteries of the brain. In the case figured in Pl. IX. 1, for instance, although there was a retinal hæmorrhage due to the effect of chronic renal disease, the cerebral symptoms pointed unmistakably to softening rather than to hæmorrhage.

Consecutive changes are very rare in senile arterial thrombosis. Optic neuritis certainly due to this cause is scarcely

ever met with. Its occurrence would not be surprising, since the secondary inflammation about an infarcted area might be adequate to cause it, but practically it is almost unknown. A case of optic neuritis, however, with and apparently due to, atheromatous softening is recorded by Wilbrand.[1] In some cases on record it is most probable that the papillitis was nephritic—a source of fallacy to be carefully borne in mind.[2] Atrophy of the discs has, in rare cases, been observed to supervene.

In some cases, however, the obstruction by thrombosis of the internal carotid may give rise to alterations in the eye, which have been hitherto observed only after death, but which must be attended by marked ophthalmoscopic changes. Such a case was described long ago by Virchow.[3] A man aged forty-six who had an attack of apoplexy, leaving right hemiplegia, died from a melanotic cancer of the liver. The internal carotid was obstructed by a thrombus, probably spontaneous, since no embolus was found, and there was fatty and calcareous degeneration of the wall of the vessel. There was a large area of softening in the left hemisphere. The ophthalmic artery was patent, evidently by a collateral circulation having been set up. The vitreous was transparent, the retina thickened, and around the papilla were four opaque white spots, which were, however, found to be due to the persistence of the medullary sheath of the nerve fibres. The ganglion cells were granular. The elements of the

[1] "Arch. für Ophth.," Bd. xxxi. p. 119, Pl. 3.

[2] A case is recorded by Wurst, for instance (Virchow's "Jahresbericht," 1877, ii. 463, from the "Przeglad lekarski"), in which optic neuritis, "stauungs-papille," was associated with cerebral softening—a spot the size of a walnut in the posterior portion of the left hemisphere, and a second, the size of a bean, in the pons Varolii. Sudden complete amaurosis had come on a few days before. There was, however, interstitial nephritis and hypertrophy of the heart, and it is most probable that the optic neuritis was due to the renal disease. In the remarkable case figured in Pl. VI. 2, optic neuritis coexisted with softening from extensive arterial disease, the results of old traumatic meningitis, but inflammatory (?) growths existed beneath two old fractures of the skull. The man had had syphilis, but the lesions presented no syphilitic character.

[3] "Arch. für Path. Anat.," Bd. x. 1856, p. 189.

nuclear layers showed a tendency to arrange themselves in lobular cylinders. Another case of the same character which came under my observation has been before alluded to (p. 32), and in it the ophthalmoscopic changes would probably have been much more striking. Although the origin of the ophthalmic artery was closed by clot, the central artery of the retina retained a channel, narrowed by clot formed upon its walls. Some retinal branches were pervious, others closed. The retina presented atrophy of all its structures, and was reduced to about two-thirds of its normal thickness.

It is important, therefore, to watch the fundus continuously in cases of thrombosis in the region of the internal carotid. It is probable that the obstruction of the carotid would always be accompanied by a sudden diminution in the size of the retinal artery, the degree of this, and the occurrence of parenchymatous changes in the retina, depending on the character of the anastomoses of the ophthalmic artery. These are usually abundant, chiefly with the facial, but also to a less extent with the middle meningeal.

Softening from Arterial Thrombosis due to Blood States.—In this condition, which is rare except in the puerperal state, ophthalmoscopic changes have been found only in cases of septicæmia (*q. v.*).

Softening from Venous Thrombosis.—Ophthalmoscopic changes are unknown. In thrombosis of the cavernous sinus, it is said that there may be double optic neuritis and exophthalmos. Slow obliteration of this sinus, however, may cause no ophthalmoscopic changes.

3. Primary Softening.—Primary softening of the brain is still a region of cerebral pathology of which we know little. Apparently three forms occur, acute and subacute inflammatory softening, and a senile form of chronic softening.

Inflammatory Softening.—The acute form is only known in connection with injuries in which meningitis is never absent, and ophthalmoscopic changes must be ascribed to this rather than to the morbid process in the brain.

Subacute Softening is a possible lesion, symptoms suggesting it being met with especially in gouty persons, but no optic neuritis has been seen in connection with it—a fact of much importance, since it is upon this and upon the occasional retrogression of the symptoms that the hypothetical diagnosis has chiefly rested.

Chronic Softening is a certain senile lesion, but is extremely rare. A few cases have been described, but in this the nature of the malady has not been suspected during life. Apparently it is not attended with ophthalmoscopic changes.

Abscess of Brain.

The only changes known are consecutive. Optic neuritis, which differs in no respect from that due to cerebral tumour, is found in many cases: the papillæ are swollen, red, and opaque, the vessels concealed, and hæmorrhages may be present. But the neuritis is frequently absent; the rapidity with which the abscess develops or increases being, apparently, the chief element in the disease on which the presence of neuritis depends. This element, however, is merely the result of the intensity of the inflammation which causes the abscess, and so we trace the result to the condition which, beyond any other, seems to determine this effect of a cerebral lesion,—the amount of irritation produced by the central disease. This condition, however, it should be remembered, is subject to another—that of time. Several days are necessary for the development of neuritis; sometimes, indeed, when the morbid process is in a distant part of the brain, several weeks may be required. Hence lesions in which the irritative element is most intense often end the life of the patient before their effect on the eye can be produced.

The changes in the optic nerves do not differ from those met with in tumours, &c. Dropsy of the sheath has not often been looked for, but was found in one case (Peipers), the abscess being in the right temporal lobe.

The only conspicuous difference between the cases of

abscess with, and those without, optic neuritis, is that due to the course of the malady. In perhaps the larger proportion of the cases with neuritis the bone disease causing the abscess was the result of injury. In two instances recorded by Hughlings-Jackson this was the case. The position of the abscess has been in the temporal and posterior parts of the parietal lobes beneath the surface. In a case recorded by Benedikt it was outside the optic thalamus. Abscess in the left hemisphere of the cerebellum, in a case recorded by Pfluger,[1] caused double optic neuritis, well marked, with capillary hæmorrhages on the papilla, and large extravasations beyond its edge.

Tumours of the Brain.

A.—*GROWTHS.*

Associated Conditions.—Growths may occur in the eye, of the same nature as the growth in the brain, but such cases are not common. The disc shown in Pl. III. 4 is the left disc of a boy, whose right eye was the seat of a tubercular growth, in whose brain there was another similar growth, of which vomiting and optic neuritis were the only signs. In such a case the ocular growth becomes an important symptom. Choroidal miliary tubercles might be expected to be found occasionally in cases in which a tubercular mass exists in the brain, but they occur rather in acute general tuberculosis, while tubercular tumours of the brain are rare in that condition. Their nature is rather that of the tubercular condition that we associate with the word "scrofula." This differs in course and associations from acute tuberculosis, although presenting the same bacilli. Thus choroidal tubercles are met with far more frequently in tubercular meningitis than in the tubercular growths.

Consecutive Changes.—Optic neuritis is *the* ocular lesion in intra-cranial growths, which are, on the other hand, its most frequent causes. It is present, in various degrees, in a large

[1] "Arch. f. Ophth.," vol. xxiv. 1878, pt. 2, p. 171.

proportion of the cases of intra-cranial tumour; in what proportion cannot be determined by statistics from published cases, on account of the selection for publication on special grounds. From my own experience I should say that neuritis occurs in about four-fifths of the cases. This is a much smaller proportion than has been deduced from published cases. Annuske and Reich, for instance, collected eighty-eight cases with ophthalmoscopic examination and autopsy, and found that there was no ophthalmoscopic change in only five per cent. But these cases have all been recorded during the period when ophthalmoscopic observation possessed the interest of novelty, and a far larger proportion of cases with neuritis has probably been published than of cases without neuritis.

It does not seem possible at present to say on what the occurrence of optic neuritis depends; why it is present in the majority, absent in the minority. Position of growth has apparently no direct influence on its occurrence, and only an indirect influence, insomuch as secondary meningitis near the nerves is more considerable when the tumour is not far from that part of the base. But the influences through which neuritis is caused seem to be exerted from any situation. It has been met with in tumours of every part of the cerebral hemispheres, of the pons Varolii, the crura cerebri, the cerebellum. Tumours of the medulla below the pons usually cause death too quickly for optic neuritis to be developed; but my colleague, Dr. T. Barlow, has met with a case of neuritis from a small tumour in the middle of the medulla oblongata. Allbutt thinks that tumours of the anterior lobes are more uniformly attended with neuritis than those of other parts, but I have seen a large growth in the anterior hemisphere with normal discs throughout.

Nor does the nature of the tumour apparently influence the development of neuritis. It occurs with every variety—glioma, sarcoma, tubercle, syphiloma. The most frequent forms of tumour are those which are most usually associated with optic neuritis; and they are also those in which

neuritis is most frequently absent—syphilomata, tubercles, and gliomata. At the same time, growths that infiltrate and only damage the nerve elements late in time and little in comparison with the amount of the growth, seem to have less tendency to cause neuritis than those which damage readily. A like difference—perhaps, indeed, related—is seen from the amount of adjacent inflammation that is produced. The greater these secondary processes about the tumour, the more readily does neuritis occur. Hence the nature of a growth has an indirect, though not a direct, effect. In a case of my own, of a glioma infiltrating almost the whole of the medulla oblongata, which was under observation for two months before death, there was no optic neuritis at any time.

The size of the tumour also seems to have little influence in producing neuritis. I have twice seen syphilomata the size of half an egg without optic neuritis. One of the largest intra-cranial tumours I have met with was a sarcomatous growth, the size of the closed fist, growing from the dura mater, and compressing, not invading, the brain over the posterior portion of the parietal lobe, a tumour which must have increased the intra-cranial pressure as much as it is ever increased directly by a growth, and in this case the discs, repeatedly examined from soon after the onset of the symptoms until death, about six months later, were perfectly normal; and a similar case is fully described by Byrom Bramwell in his recent work on "Intra-cranial Tumours," pp. 11, 12. On the other hand, Benedikt has recorded a case of well-marked neuritis with much swelling and hæmorrhages, due to a tubercle of the pons Varolii no larger than a cherry. There were no signs of meningitis.

The chief facts at present known regarding the mechanism by which optic neuritis is produced have been already discussed (p. 78). Some points having special reference to tumour may be again adverted to. It is clear from the facts stated above—and a long list of similar cases might be given—that encephalic tumours do not cause neuritis by the direct effect of their mass on the intra-cranial pressure. Perhaps

no form of cerebral tumour is attended with optic neuritis in a larger proportion of cases than glioma, which commonly does not press upon, but invades, the brain substance, and often occupies the invaded tissue almost bulk for bulk.

It has been thought that the rapidity of growth of a tumour influences the occurrence of optic neuritis, but a limited experience of these cases, or a very short search among recorded cases, disposes of the hypothesis, at any rate in an absolute form. Rapidity of growth may be one factor in the production of neuritis, and an important factor in determining the rapidity or slowness of the course of the neuritis, but it certainly does not alone determine its occurrence.

There is, however, one mode in which neuritis is produced which may sometimes be distinctly traced post-mortem, viz., by the mechanism of meningitis. The disc shown in Pl. III. 3, for instance, was in a case of tumour originating in the pineal body and invading the anterior corpora quadrigemina. The changes in the disc were very gradual in development, and moderate in degree. There was no general meningitis, but the orbital lobules were gently adherent, and fine shreds of lymph were visible on the dura mater after their separation. The optic nerves in front of the commissure were swollen and reddened. Microscopical evidence of neuritis of the nerve-trunk was very distinct. In another case of tumour (glioma) of the anterior lobe, in which the neuritis was of the form most characteristic of tumour, greyish-red, with much swelling, the microscopic changes in the nerve-trunk, most intense behind the foramen, indicated a communicated descending neuritis, and old adhesions over the tumour showed that there had been local meningitis. It must be remembered that, in such cases, whatever mechanism leads to the occurrence of neuritis without meningitis may influence the degree and course of that which is set up by meningitis.

In most cases optic neuritis is a transient event in the history of a cerebral tumour, not a constantly-associated condition. A tumour may exist and cause symptoms for a

considerable time without leading to any change in the eyes, and then optic neuritis may be rapidly developed, run its course, and pass away, in many cases leaving atrophy of the discs, while the symptoms of the tumour continue or increase for months or years. It is not only that a tumour takes a certain time to cause optic neuritis, but it often exists for a considerable time before the mechanism for the production of neuritis, whatever that may be, is set in operation. A tumour may exist and cause symptoms for years before optic neuritis is produced. A striking instance of this is afforded by a case which was under the care of Dr. Hughlings-Jackson, who had examined the eyes repeatedly during nine months, and always found them normal. Then neuritis came on, but subsided, and in six weeks the discs were again normal, and continued so till death. The microscopical appearance of part of the papilla is shown in Fig. 22, p. 63. Dr. Jackson has recorded[1] a still more significant case, in which a man had had symptoms of cerebral tumour for nine years: during the last three years his discs had been repeatedly examined and found normal. Six weeks before death neuritis was discovered.

In many cases in which neuritis occurs long after the symptoms of tumour have existed, its occurrence precedes death by no long interval.

The appearance of the discs in intra-cranial tumour is that of neuritis in its most typical form, as described in a preceding page (p. 49). The neuritis may stop at one or another of its stages, constituting what may be termed varieties of neuritis. As already stated, until our knowledge of the relation of the appearances to their causes is much more extensive, and founded on more minute and full observation of the conditions of origin, macroscopic and microscopic, a division of neuritis into varieties according to its degree is much more useful than a separation of forms according to hypothetical modes of origin. Those varieties or stages have been already enumerated (p. 93). Each of the earlier stages may or may not be accompanied by obvious

[1] "Med. Times and Gazette," Sept. 4, 1875.

over-distension of veins, and each may be accompanied by extravasations.

The neuritis of tumour is in most cases double, sometimes equally advanced in the two eyes, often more intense and subsiding earlier in one than in the other. Rarely the affection of the disc is unilateral, and this, although the tumour may be in the brain, where growths commonly cause double neuritis. In two cases of this character recorded by Hughlings-Jackson,[1] and in one described by Field,[2] the neuritis was on the side opposite to the tumour. In one recorded by Greenfield,[3] however, where unilateral neuritis accompanied an abscess in the top of the temporo-sphenoidal lobe, the neuritis was on the same side as the lesion. Possibly the inflammatory process extended to the nerve as it passed to the optic foramen.

Symptoms.—The symptoms of the neuritis which accompanies cerebral tumour have been already fully described (p. 69). It must be remembered that all symptoms may be absent, the acuity of vision, the fields of vision, and colour-vision may also be unaffected, as in many of the cases figured in the plates and referred to in the description of the symptoms of neuritis. It must also be remembered that affections of sight of various kinds may co-exist with neuritis, and be due, not to the intra-ocular, but to the intra-cranial disease.

Regarding the course of the neuritis in cerebral tumour, it is important to note that the neuritis often coincides at its onset with an obvious increase in the other symptoms of the cerebral tumour. This was pointed out, long ago, by Dr. Hughlings-Jackson. Instances of it are frequent, but at the same time exceptions are not rare. It is probably true, however, that *the occurrence of optic neuritis indicates progress in the morbid growth and its consequences.*

With regard to the course of the neuritis, it is necessary to distinguish two classes of cases. One of these is where the

[1] "Ophth. Hosp. Rep.," 1871, and "Brit. Med. Journal," July 20, 1872.
[2] "Brain," July, 1881, p. 247.
[3] "Brit. Med. Journal," 1886, p. 317.

progress of the tumour, either spontaneously, or under the influence of treatment, becomes lessened or arrested after the onset of the neuritis; the other, where the progress of the tumour to which the neuritis is due is uninterrupted.

In the first event, the neuritis commonly subsides. It may pass away completely, even although it has reached the stage of considerable swelling and obscuration of disc and vessels, with distended veins and narrowed arteries, and sight may throughout be unimpaired. This occurred, for instance, in the cases shown in Pl. IV. 1, 2, 3, 4, V. 3. Or, less commonly, a slight or moderate damage to sight, from the inflammatory swelling and damage to nerve fibres, may pass away. Very frequently, however, although the neuritis subsides, amblyopia occurs or increases when the nerve fibres suffer from compression from the contracting tissue. The last is the more likely to occur the longer the neuritis has lasted, because there is then more tissue formed, incapable of removal.

Instances of each course are often seen in syphilitic tumours, and not rarely where there is strong reason to believe that a scrofulous tumour exists—a cerebral or cerebellar tubercle. In cases in which the neuritis is slight and commencing, a subsidence of the neuritis may be the first sign of the improvement. It was so in the case figured in Pl. V. 4, in which the neuritis passed away before there was any improvement in the symptoms, and then slowly the paralysis lessened, and improved up to a certain point, at which it became stationary, no doubt from the tumour (probably tubercular) ceasing to grow, and becoming, from partial degeneration, smaller, and thus permitting damaged tissue near it to recover, while the destruction, which had before taken place, persisted. In syphilitic tumours, arrest can be obtained much more rapidly than in tubercular growths, and a considerable neuritis may pass away without damage to vision (Pl. IV. 1 & 2, 3 & 4, VI. 4 & 5). In these cases, however, if a considerable neuritis exists before the treatment affects the tumour, tissue-changes too often progress in the disc to an extent which leads to loss of sight

even though ultimately the cerebral lesion ceases to increase and becomes quiescent (Pl. IV. 5, 6). Occasionally, although rarely, an analogous arrest of growth occurs in other tumours, attended with degeneration and calcification. The neuritis may, in these cases, subside with the change in the growth.

In the cases in which the tumour causing the optic change continues its growth, as most tumours of other descriptions than the tubercular and syphilitic growths, the course of the neuritis differs according to the intensity of the inflammation. When this is considerable, the neuritis remains for a time at its height; commonly the signs of strangulation are developed, and then the neuritis subsides slowly into atrophy. The inflammation, as it were, terminates itself, and its consequences remain. When the neuritis does not reach so intense a grade it has a much longer duration. The lilac-grey neuritis, with little sign of strangulation, may persist for months without much change, and then slowly subside to atrophy; sight perhaps being little damaged until the subsidence, when the tissue formed during the long duration of the inflammation compresses the nerve-fibres. In a still slighter degree, that of "slight neuritis," for instance (p. 93), the change may persist without alteration for a very long time. In the case represented in Pl. V. Figs. 1 and 2, the appearance of the discs was unchanged for a year and a half, and when the patient was again seen a year later, the neuritis was nearly in the same degree, although the least inflamed portion of the disc had become grey and sight was gone.

There is at present little direct information regarding the conditions which determine the course and duration of neuritis in the cases in which the cerebral tumour continues its progress. But it has been seen that the onset of neuritis may accompany, or succeed, an increase in the symptoms due to the tumour, such as indicates an increase in the size or irritative action of the growth itself. And we have seen also that the early subsidence of neuritis may attend a diminution in the other effects of the tumour such as may

be taken as indicative of an arrest of growth, or even a diminution in size. These facts taken together indicate that the course of the neuritis is, to some extent at least, dependent on, and influenced by, the course of the tumour. This conclusion is corroborated by the fact that in some cases of tumour of very chronic nature, the course of the neuritis is equally chronic. The case mentioned above (Pl. V. 1 & 2) is a striking illustration of this, since the progress of the very marked symptoms was but slight during the year and a half, in which the neuritis was absolutely stationary. In rare cases, as in that recorded by Field and above referred to, in which, without retrogression of the tumour, neuritis subsides without influencing vision, the affection of the optic nerve is probably largely due to excessive secondary effects of the growth. In this case there was adjacent softening out of all proportion to the size of the growth itself.

Significance.—The value of optic neuritis as an indication of the existence of an intra-cranial tumour is very great. Tumour is the cause of the majority of cases of neuritis due to intra-cranial disease. On the other hand, neuritis is present, at some period, in at least four-fifths of the cases of tumour, and it may be the only unequivocal sign of the organic intra-cranial disease.

It is important to remember that the neuritis is a transient condition, however long its duration, and that its effects continue a much longer time than the inflammation. The atrophy left by neuritis may constitute unequivocal evidence of the antecedent inflammation, and where actual atrophy is not left, the state of the disc and the narrowing of the vessels may show clearly that there has been previous neuritis. Unfortunately it is not always possible, in old-standing cases, to say from the aspect of the discs how the atrophy originated. If the neuritis was moderate, and the adjacent choroid undisturbed, a clean cut disc may be left, and the narrowing of the vessels may not be greater than is sometimes seen in cases of atrophy of the disc of other forms. The concealment of the lamina cribrosa is, however, usually complete. Valuable information may also be gained

from the circumstances under which the loss of sight came on; the existence at the time of cerebral symptoms makes it probable that the atrophy was due to neuritis.

It is not only during life that neuritis may assist the diagnosis of tumour. As an instance, I may mention the case of a man who died soon after his admission, with hemiplegia, into University College Hospital. The autopsy revealed a soft area, bounded and crossed by trabeculæ of firmer tissue, which was at first thought to be an area of old softening with some connective-tissue formation in and about it. It was suggested, however, that it might be a tumour. Before it was examined with the microscope, the backs of the eyes were removed, and found to present distinct evidence of neuritis—swollen papillæ with hæmorrhages. A diagnosis of probable tumour was therefore made, and was fully confirmed by the microscope.

From the facts given above it is evident that optic neuritis may, in some cases, afford not only diagnostic, but prognostic indications. A subsidence of neuritis which has not reached any considerable degree of intensity, may be taken as indicating, in most cases, a retrogression of the growth, and a neuritis of very chronic course affords evidence that the progress of the tumour is equally chronic. It might be supposed, therefore, that the absence of neuritis would indicate still greater chronicity. This, however, cannot be inferred, since tumours of very rapid course may be unattended with neuritis, and it is only when neuritis is actually present that a prognostic inference can be drawn.

It has been remarked that optic neuritis in tumour of slow growth often occurs not long before death. In such cases, also, it affords some prognostic indication. In more acute cases, or in those in which it developes early, it has not the same significance. It would appear as if the mechanism for the production of neuritis were, in the latter cases, readily excited, while in the former it is the result of changes of such a degree as to be incompatible with the long continuance of life.

Simple atrophy of the optic nerves also results from intracranial tumours, but only by the mechanism of compression of the fibres of the optic nerve where all those proceeding from one eye or both can be destroyed. Thus, such atrophy only occurs when there is pressure on the chiasma, or on one of the nerves in front of the chiasma. Pressure on one tract seldom causes sufficient alteration in the disc to be attended with more than slight pallor and slight shrinking in both eyes. Theoretically, pressure on both tracts should cause conspicuous atrophy, but no instance is known; perhaps life, in such a case, is not prolonged for the time necessary to permit visible alteration. The simple atrophy is thus "secondary" in nature, and due to the direct effect of the growth on the fibres of the nerves, and also to the secondary consequences of the tumour—especially the pressure of ventricular effusion on the chiasma. It is doubtful whether this form of atrophy ever results from the damage to the nerve by inflammation, such as may be produced by a secondary meningitis. In tumours, the tendency for a communicated inflammation to spread down the nerve is so strong that optic neuritis seems to be invariable. But the visible inflammation is often slighter than the failure of sight, and the ultimate atrophy may be in part simple although apparently papillitic. Such atrophy has the characters of secondary atrophy of the optic nerves, the features and origin of which have been already described. Great caution is also necessary in inferring, from the appearance of discs long after the onset of the atrophy, that this was simple and not neuritic. We have already seen that the characters of the latter may ultimately resemble very closely those of the former. Moreover, not only may there be a combination of the two processes (secondary atrophy from greater damage near the chiasma, and the atrophy from papillitis), but the two may occur at different periods. The chiasma may be compressed by ventricular effusion, or even by a fresh increase in the tumour, *after* neuritis has gone on to partial atrophy. Sight, damaged much or little by the neuritic process, may fail rapidly at a

subsequent period from secondary pressure effects. This was well illustrated in the case of a man who was admitted with double optic neuritis, impairment of vision, and symptoms pointing to a tumour of the base involving the ocular nerves. Under treatment with iodide, the neuritis quite subsided, and vision improved until it became almost normal, with perfect fields. Nine months later, however, deterioration of sight again occurred, the fields remaining normal, but nothing could be detected with the ophthalmoscope. Six months after the relapse he had a fœtid smell in the left nostril and loss of vision in the temporal half of the right field. A few months later there was failure of sight in the temporal half of the left field, and there were also indications of pressure on the right fifth and left third nerves. He was under observation for five years later, during which time most of the symptoms passed off. There was, however, atrophy of both discs, with qualitative perception of light only, and complete loss of the sense of smell. There must, in this case, have been a tumour at the base of the brain, pressing on the anterior part of the chiasma, and also involving the nerves mentioned.

B.—*HYDATID CYSTS.*

Associated Changes.—A cysticercus has been occasionally observed in the vitreous humour, but the coincidence of a parasite in the eye with symptoms of cerebral tumour due to another in the brain, has not, I believe, hitherto been recorded.

Consecutive Changes.—Optic neuritis is frequent in cases of hydatid disease of the brain, and has all the characters of the neuritis which occurs in growths—swollen papilla, obscured and tortuous vessels, hæmorrhages. It has been observed with hydatid cyst of both cerebrum and cerebellum. It may go on to consecutive atrophy, life being prolonged for years. The few cases on record of neuritis associated with cysts in the brain, the nature of which was not ascertained, were probably examples of hydatid disease.

Labio-Glossal Paralysis.

In chronic bulbar paralysis, due to degeneration, ophthalmoscopic changes are extremely rare. Unilateral atrophy was once seen by Galezowski, and Robin quotes a case from Dianaux of rapid atrophy of both nerves in the course of the affection in a man aged sixty-seven. It was accompanied by transient paralysis of one sixth nerve. Sight was lost completely in two months, but considerable subsequent restoration of vision (up to $\frac{1}{10}$) occurred.

Intra-Cranial Aneurism.

Miliary aneurisms have been spoken of in connection with cerebral hæmorrhage. Intra-cranial aneurisms of larger size are not, as a rule, accompanied by any *associated* ocular changes: those of the central artery of the retina being too rare to be of significance. Nor do they often cause *consecutive* changes, unless their position is such as to press upon the optic nerve (causing unilateral amaurosis and secondary atrophy), on the chiasma (bilateral atrophy), or, very rarely, on the optic tract (causing hemianopia). An aneurism of the internal carotid may obstruct the cavernous sinus, and cause transient distension of the retinal veins, without papillary changes, but the pressure is relieved by the free communication of the ophthalmic and facial veins ; the enlarged angular vein may be conspicuous beneath the skin. In rare cases, however, an aneurism in this situation has led to optic neuritis, as in a case recorded by Michel;[1] double neuritis, with evidence of obstruction, was the first sign of a cirsoid aneurism of the two internal carotids. It pressed on the optic nerves at the spot, and these showed evidence of interstitial inflammation. Holmes of Chicago has recorded several cases in which optic neuritis co-existed with intra-cranial bruit, and in the only one on which a post-mortem was obtained an

[1] "Arch. f. Ophth.," xxxiii. 2, p. 225.

aneurism of the internal carotid was found; but there was also an adjacent growth in the pituitary body.

In an interesting case[1] (by Jeaffreson of Newcastle-on-Tyne), although there was no post-mortem examination, an aneurism of the internal carotid was most probable, and caused unilateral papillitis. A loud intra-cranial murmur could be arrested by compression of the carotid; there was paralysis of the third nerve, and subsequently aphasia developed.

The origin of the papillitis in these cases is probably a descending inflammation, extending to the nerve from that which always exists around an aneurism. That the papillitis is not the effect of compression of the cavernous sinus is probable from the fact that aneurisms which produce the same effect on the sinus may or may not be accompanied by papillitis. Moreover, when there is papillitis the enlarged communications with the facial vein may (as in Jeaffreson's case) afford the same evidence of relief to mechanical obstruction, which is supposed to prevent the papillitis (when this is absent) by those who ascribe it to the mechanical influence alone.

Internal Hydrocephalus.

Simple internal hydrocephalus, without a growth, is not at first attended by ophthalmoscopic changes unless the state is due to inherited syphilis. They may be absent throughout, even though the distension of the ventricles is such as to cause a marked increase in the size of the head. Sometimes there is slight fulness of the retinal veins. Sight often fails at a later period, and in some cases early, and the signs of simple white atrophy of the optic nerve are then present. In several cases the onset of the atrophy has been watched, and the occurrence of any neuritic process excluded. In a few cases the atrophy has been preceded by signs of neuritis similar to that seen in tumour; it is usually slight in degree, but was considerable in a case recorded by Wildbrand and Binswanger.[2]

[1] "The Lancet," March 8, 1879.

[2] "Centralbl. f. med. Wiss.," 1879, p. 923.

The simple atrophy of the nerves is usually due to the pressure of the distended third ventricle on the optic chiasma. In one adult case, mentioned by Förster, the distended ventricle appeared at the base of the brain as a bladder measuring ten lines by eight.

It has been said by Bouchut that the ophthalmoscopic changes may serve to distinguish chronic hydrocephalus from the large head of rickets; but, owing to the lateness of the optic changes, the cases must be very rare in which the nature of the disease is not distinct long before ophthalmoscopic signs are present.

DISEASES OF THE MEMBRANES OF THE BRAIN.

Meningeal Growths.

Tumours springing from the pia mater always involve the cerebral substance to a greater or less extent, either by invasion or compression, and their effects have been included in the account of the cerebral tumours.

Tumours springing from the dura mater differ in their effects according to two characteristics—first, their tendency to invade; secondly, their position, whether at the base of the brain or on the convexity. They commonly cause the same effects, in the brain and on the eye, as growths in the brain itself.

Growths springing from the dura mater of the base of the brain cause optic neuritis much more frequently. When in the front of the base, the inflammation around the growth may extend directly to the nerve. But when more distant, as in the posterior fossa, optic neuritis is still a frequent consequence and is often intense, even when the nerve centres are not invaded. Those that invade the brain have the same tendency to cause optic neuritis as tumours beginning in the brain substance. But the compressing growths have this tendency in far slighter degree, and it is less the slower the growth of the tumour. The more rapidly the

pressure is induced, the greater and more acute is the secondary inflammatory process in the compressed part, manifested by its softening. With very slowly growing tumours such softening may be entirely absent, and the tendency to the occurrence of optic neuritis is very much slighter. I have seen a tumour the size of the closed fist, which had compressed the hinder half of one hemisphere so as to produce a depression corresponding to the growth, in which there was no optic neuritis up to the end, and no sign that optic neuritis had ever existed. Hence the absence of optic neuritis is evidence of some value that a tumour at the surface of the brain springs from the membranes and is not invasive. Cases are on record, moreover, in which the optic neuritis was for a long time the only symptom of such a growth; as in one case in which, after the neuritis had existed for months, hemiplegia came on, and was found to be due to a sarcoma springing from the periosteal dura mater, and which had compressed the left hemisphere of the cerebellum and the left side of the pons Varolii.[1] In the case figured in Pl. V. 5, optic neuritis, although not the earliest symptom, reached its height before any motor paralysis occurred. The tumour sprang from the dura mater, and had compressed the right side of the pons and right hemisphere of the cerebellum.

In some of these cases secondary meningitis may be traced along the base of the brain. Such inflammation is produced by meningeal growths even more frequently than it is by tumours in the substance of the brain, and it may play an important part in the production of the changes in the eye.

Meningitis.

The effects of meningitis on the eye vary much according to its seat, being slight and late when the inflammation is at the convexity of the hemisphere, considerable and

[1] Pagenstecher and Genth's "Atlas of the Path. Anat. of the Eyeball," Pl. xxxiv. Fig. 3.

early when the meningitis is at the base. In some cases, especially of the former class, ophthalmoscopic changes are entirely absent, and when present they attend the stage of developed inflammation rather than the initial vascular disturbance. They thus afford, as Manz and others have pointed out, little support to the doctrine that the intra-ocular circulation shares and reveals disturbances of the encephalic vessels. It will be convenient to consider separately the changes in the several forms of meningitis.

Simple Meningitis.—Acute simple meningitis of the convexity is usually unaccompanied by ophthalmoscopic changes; only when it has lasted for a considerable time is neuritis sometimes developed. In a case of purulent meningitis, suppurative inflammation of the eye (chemosis and post-mortem infiltration of the retina with pus) was observed by Berthold,[1] but was probably coincident. Leube[2] has recorded a case of purulent meningitis of the convexity secondary to septicæmia in which there was intense inflammation of the optic nerve in front of the commissure. The only changes in the eye were distension of the retinal veins and hæmorrhages. I have seen well-marked neuritis in a case of septic meningitis (post-puerperal) with grave cerebral symptoms. The patient recovered.

Chronic simple meningitis of the convexity, slight in degree (such as that of which traces are often found in the brains of drunkards), is also commonly unattended by any optic change. The slight œdema and congestion of the disc, sometimes seen in chronic alcoholism, is probably the result of the toxæmic condition rather than of the encephalic change.

Simple meningitis of the base is rare, except in association with tumour or some bone disease. Optic neuritis may occur by direct propagation, and in those cases in which the disease is chronic, the visible changes in the disc may be

[1] "Arch. f. Ophth.," Bd. xvii. 1874.

[2] "Deut. Arch. f. klin. Med.," 1878, xxii. 263.

considerable in degree and duration. Basilar meningitis is, however, in most cases tubercular or syphilitic.

TUBERCULAR MENINGITIS: *Associated Condition.*—Tubercles of the choroid may now and then be found in tubercular meningitis, and furnish valuable diagnostic information. But they are less frequent, as Cohnheim pointed out, in tubercular meningitis than in general tuberculosis without meningitis. Heinzel[1] never saw them in forty-one cases of tubercular meningitis which he examined with the ophthalmoscope, and the case figured (Fig. 49) was the sole instance in which they were found in twenty-six cases examined by Garlick at the Hospital for Sick Children. The few recorded cases in which neuritis due to meningitis co-existed with tubercles of the choroid have been collected by Brückner.[2]

Consecutive Changes.—A peculiar marbled reflection from the retina has been described by Leber and Hock, occurring especially in the neighbourhood of the veins. They have seen it in conjunction with tubercles of the choroid, and state that it is not due to neuritis occurring earlier. Nevertheless, redness of the disc is sometimes observed in association with this condition.[3] A somewhat similar reflection, chiefly around the disc, has been described by Manz as the most frequent change. He associates it with œdema of the sheath of the optic nerve, and it may be due to a slight œdema of the retina (compare Pl. I. 3).

Changes in the optic discs of more considerable degree are, however, present in tubercular meningitis in such a proportion of the cases as to constitute a very important symptom of the disease. The frequency of the occurrence has been variously stated. The discs are often normal throughout in the rare cases in which the tubercular

[1] "Jahrbuch für Kinderheilkunde," 1875, p. 334.

[2] "Arch. f. Ophthal.," vol. xxvi. pt. 3, 1880, p. 154.

[3] It is doubtful whether this appearance is really pathological; a condition very like it is met with apart from disease, often called the "watered-silk retina." For an explanation of this appearance, see Gunn, "Ophth. Hosp. Rep.," vol. xi. p. 348.

inflammation is confined to the convexity of the brain. In some cases of basal meningitis, also, changes are entirely absent. Garlick,[1] of twenty-six cases carefully watched at the Children's Hospital, found the discs normal throughout in five; distinct swelling was developed in about half the whole number, increased redness only in one quarter, and in a few others only distension of veins. In many of these cases, however, the changes were slight, and their pathological character was recognizable only by their development under observation. It is probable, then, that considerable changes are present in one-half the cases, and that in two-thirds of the remainder slight alterations will be found, if the discs are watched with care from day to day. The occurrence of congestion and œdema of the disc seems to be especially related to the occurrence of inflammation, and the formation of lymph, in the anterior part of the base, about the chiasma and the optic nerves.

The degree of change is rarely great. The disc becomes full-coloured, and its outlines hazy. Sometimes this and distended veins constitute the only morbid appearance. More often swelling, with undue striation, becomes visible on direct examination, and the edges of the disc gradually cease to be recognizable. The disc has sometimes a reddish-grey aspect. In several recent cases I have noted that the colour of the swollen papillæ was much paler, especially on examination by the indirect method, than in the early stage of the acute neuritis of cerebral tumour; the aspect suggesting the idea of a subsiding neuritis rather than one that is commencing, and this in cases in which the neuritis was quite recent. The neuritis rarely passes into a more intense degree, perhaps because life is only prolonged sufficiently in cases in which the inflammation is not intense. The veins are often, though not always, over-distended from the first. In Garlick's observations their distension was especially related to excess of subarachnoid fluid; when the quantity of this was normal, there was no distension of the sheath—a fact of much importance. Occasionally white lines along

[1] "Med.-Chir. Trans," 1879, p. 441.

the sides of the vessels are unduly conspicuous. Hæmorrhages are rare.

Sometimes white spots are seen in the neighbourhood of the swollen disc. They are in the substance of the retina, and consist of an accumulation of lymphoid corpuscles in the nuclear and molecular layers, or of degeneration of nerve-fibres. They may readily be mistaken for tubercles of the choroid. It has been thought that they are of the nature of tubercles, and they have accordingly been described as retinal tubercles, but very similar spots are seen in neuro-retinitis from other causes. Occasionally a gauze-like opacity is seen over a wide area of the retina, with scattered white points and flakes (Heinzel). Very rarely retinal hæmorrhages are associated with the papillitis.[1]

The changes that occur in tubercular meningitis are always double, though often more advanced on one side than on the other. In some cases the excess was found by Garlick to be on the side of the chief cerebral change, but in a few it was on the other side. In most cases the patients die not long after its development, and sight suffers little. In the rare cases that recover the inflammation does not become intense within either the skull or the eye. In such cases the optic neuritis is of extreme diagnostic importance. As the cerebral symptoms subside, the neuritis passes away, and sight is preserved or restored. This has been pointed out by Clifford Allbutt, and two probable instances are described by Garlick. The symptoms were headache, vomiting, constipation, irregular pulse, normal temperature, and the development of ophthalmoscopic changes under observation. In both cases recovery was complete. In another case observed by him an increase in pulmonary symptoms was attended by a marked decrease in the cerebral symptoms, and in the optic changes, for five days before death.

Cases of optic nerve atrophy of old-standing are occasionally seen in which sight was lost in early life with acute cerebral symptoms very like those of an attack of tuber-

[1] Heinzel, loc. cit. p. 341, Cases 6, 16, 19, 26.

cular meningitis. Several such cases have been related by Hutchinson.[1] Incipient atrophy was noted by Heinzel in one case of long duration, and in two others he observed the initial stage of consecutive atrophy. In some of the cases of recovery from supposed tubercular meningitis with ophthalmoscopic changes, the symptoms, it must be remembered, may possibly have been due to a tubercular mass in the brain. The symptoms of such a tumour sometimes resemble closely those of tubercular meningitis, but much more frequently pass away.

The neuritis which accompanies tubercular meningitis was regarded by v. Graefe as affording the typical example of descending neuritis, the inflammation passing directly from the membranes to the optic nerves. With this my own experience accords. In some cases the existence of inflammation in the trunk of the nerve is obvious on naked-eye examination. The nerve is swollen, softened, and reddened. In most cases the descending neuritis may be demonstrated by microscopical examination.

Besides the distension of the sheath, which sometimes, but not always, coincides (and has been supposed to be the cause of the neuritis), more pronounced lesions are often found in it. The sheath usually presents, under the microscope, evidences of inflammation and exudation, which were found by v. Ziemssen[2] to extend from the chiasma to the eye. Moreover, Michel,[3] in a case in which there was a cloudy halo around the papilla, found not only effusion into the sheath, but numerous miliary tubercles in both the dural sheath and pial tissue.

In a considerable number of cases the symptoms of meningitis are distinct before the ocular changes are developed. In such cases the ophthalmoscope corroborates rather than assists the diagnosis. But in some cases the cerebral symptoms are latent or dubious, and in these the examination of the eyes may afford very valuable help, and it is probable

1 "Ophth. Hosp. Rep.," v. 310 and ix. 124.
2 "Jahrb. f. Ophthalmologie," 1878, p. 242.
3 "Deutsch. Archiv. f. klin. Med.," xxxii. p. 439.

that it would do so in at least one-third of the cases. Of the twenty-six cases watched by Garlick, the ophthalmoscope was of real diagnostic assistance in six, and would doubtless have been so in a larger number had earlier examination been practicable. In one case, which lasted twenty-six days, the other symptoms were indefinite until the nineteenth day, but on the fourteenth day the ophthalmoscopic changes were so unmistakable that the diagnosis of meningitis was confidently made. In another case, ophthalmoscopic changes were distinct on the ninth day, the symptoms were diagnostic only on the fifteenth day, the patient dying on the twentieth day. In both cases, the changes about the optic commissure were much more marked than those elsewhere.

During the course of meningitis a diminution of the cerebral symptoms may be accompanied by a diminution in the ocular changes.

Syphilitic Meningitis.—Syphilitic meningitis (1) may be associated with the ocular signs of syphilis, and (2) may cause optic neuritis. When at the base, the ophthalmoscopic signs are similar to those of tubercular meningitis, but more chronic in course and more considerable in degree. When localized in the convexity, ocular symptoms may be entirely absent. If the case is not subjected to proper treatment, and local chronic meningitis persists, it is probable that the disc sometimes passes into a condition of intense neuritis, similar to that which is seen in cerebral tumour. Syphilitic meningitis is a malady about which, however, we still have much to learn. Its diagnosis from gummata is only possible by the more extensive symptoms, and a growth can never be excluded if focal symptoms are produced. Moreover, the two processes pass one into the other.

Hæmorrhagic Pachymeningitis (Hæmatoma of the Dura Mater).—According to Fürstner,[1] there may be mechanical congestion of the retinal veins and papillitis,

[1] "Arch. f. Psychiatrie," vol. viii. pt. 1.

accompanied by distension of the optic sheath with dark-coloured fluid.

Cerebro-Spinal Meningitis.—In epidemic cerebro-spinal meningitis, optic neuritis may occur, but is rare. Schirmer found it in one only of twenty-seven cases examined. Von Ziemssen[1] observed slight neuritis in one case, and in another a pale fundus with broad and tortuous veins, narrow arteries, and hæmorrhages beside the disc; at a later period white points appeared in the retina. Cyclitis and retinitis were found by Oeller.[2] Many of the retinal veins contained thrombi and granular plugs; no direct connection with the intra-cranial process could be traced. A purulent irido-choroiditis is the most frequent change in this disease.

In the sporadic (possibly rheumatic) form of cerebro-spinal meningitis, optic neuritis may occur, and may lead to atrophy. Thus Mr. E. Pope of Tring recently showed me a lad who, after a severe wetting, had suffered from intense headache, delirium, fever, and retraction of the head. Sight failed ten days after the onset. The symptoms subsided at the end of six weeks, but he remained blind, and when I saw him, six months later, there was slight perception of light in one eye only. The optic discs had all the appearance of consecutive atrophy, the centres were filled in with new tissue, the vessels narrowed, and the adjacent choroid disturbed. Such a case, however, is perhaps to be separated from most sporadic cases, since in these a conspicuous exciting cause is seldom to be traced.

Traumatic Meningitis often causes ophthalmoscopic changes, of which an instance is shown in Pl. III. 5, a case in which fever, delirium, and convulsions succeeded a fall on the head. The neuritis subsided with the symptoms. These cases are considered in the section—"Injuries to the Head." The ophthalmoscopic changes are frequent and are of the highest importance in the many cases in which other symptoms are subjective only, and when the grave

1 "Jahrb. f. Ophthalmologie," 1878, p. 243.
2 "Arch. f. Augenkrank.," vol. viii. 1878, p. 357.

nature of the effects of the injury may be doubted or even denied by those whose interests are opposed.

Diseases of the Cranial Bones.

Caries.—In caries of the sphenoid bone, or suppuration beneath the periosteum, the inflammation may extend to the optic nerve, damaging it, and causing secondary atrophy, or, descending the nerve, may produce intra-ocular neuritis. The disc shown in Pl. III. 2 is an illustration of this effect. The case was one of caries of the body of the sphenoid bone in a girl aged sixteen. There was well-marked neuritis in the left eye, but for a month afterwards the right eye was normal. Coincidently with an increase of the symptoms of meningitis, this also became inflamed, and she died a few days later. The autopsy showed caries of the sphenoid, chronic meningitis around the left sphenoidal fissure, involving the sheath of the left optic nerve. There was also general acute purulent meningitis, which had, no doubt, been the cause of the neuritis in the right eye. The damage to the nerve was just in front of the chiasma; the neuritis coincided in onset with an increase in local symptoms, which ended in an attack of meningitis, from which the patient died. When the drawing was made, the neuritis was confined to the eye corresponding to the damaged nerve; soon after the onset of the meningitis, a day or two later, similar neuritis made its appearance in the other eye. In this case there was no change in the sheath of the nerve. In a case recorded by Horner, of caries of the sphenoid, the sheath of the optic nerve was distended by purulent material as far as the eyeball.

Caries of the bone, at a distance from the optic nerves, does not cause ophthalmoscopic changes unless it excites meningitis or cerebral abscess. To this, however, an exception must be made in regard to disease of the bones of the ear, which there is reason to believe may cause optic neuritis when no abscess or meningitis is to be found. It has been suggested by Mr. Arthur Barker that the papillitis in these

cases may be the result of a septic inflammation in the middle ear, infecting directly the adjacent carotid canal, and extending along the lymphatics of the latter to the sheath of the optic nerve. Cases such as he has observed are certainly of much clinical importance, and deserve close attention on the part of the pathologist.

Thickening of the Cranial Bones.—General thickening of the cranial bones may cause optic neuritis and consecutive atrophy. Neuritis with great swelling of the papilla, was present in a case of this description in the Queen Square Hospital under the care of Dr. Buzzard. The general thickening of the bones of the skull appeared to be of a sub-inflammatory character. There was no post-mortem examination, as the patient recovered; but Michel has recorded the case of a boy who was blinded by neuritis and consecutive atrophy early in life, and who died aged fifteen. The necropsy revealed great hyperostosis of the bones of the skull, by which both optic foramina were considerably narrowed. The optic nerves were atrophied from the chiasma to the eye, but the orbital portion was greatly thickened by hyperplasia of the cellular tissue in the subvaginal space. A similar case has been described by Manz, in which the tissue between the sheath and the nerve had a semi-gelatinous aspect. Michel explains this change, by assuming that the narrowing of the foramen leads to retention within the sheath of lymphatic fluids, which cause irritation.

In other cases, similar conditions of bone, exostoses, &c., narrowing the optic foramen, have caused only simple atrophy of the optic nerve.

Diseases of the Orbit.

Inflammatory Processes in the Orbit, e.g., cellulitis (as in facial erysipelas), inflammation at the back of the orbit, or periosteal affections in which the symptoms and their course point clearly to the seat and nature of the lesion, although the pathological inference is still as unconfirmed by *post-mortem* evidence as is the case with the analogous inflammation of

the facial nerve, frequently damage the optic nerve. This damage always involves inflammation, which may or may not be seen in the papilla. The difference depends partly on its tendency to spread down the nerve, coupled with its proximity to the eye or distance from it, and partly, perhaps, on the compression of the vein. But whether there is neuritis or not, atrophy is subsequently visible, proportioned, in degree, to the impairment of sight. This may or may not have the aspect of "consecutive atrophy." There may be neuritis and its effects, but the affection of sight may be due chiefly, not to the visible inflammation, but to the changes behind the eye, at the spot primarily diseased. Hence care must be taken (as pointed out in the account of secondary atrophy) not to regard the papillitis as the chief cause of a failure of sight that may occur without any intra-ocular inflammation. It is of practical importance to remember that no forecast can be drawn from the visible inflammation—that the absence of this affords no ground for a good prognosis. In the one case, the nerve is simply compressed by the inflammatory products, or, if inflamed, the inflammation is localized. Sight is lost sometimes very rapidly, and simple secondary atrophy of the lower portion of the nerve results, occasionally with ultimate narrowing of the vessels (Allbutt and Teale). In the other case, the inflammation is communicated to the nerve, and descends along it to the eye, or inflammatory processes in the sheath lead to a secondary papillitis. In many cases the eyeball becomes prominent, usually only in slight degree, and the absence of such prominence is of no negative significance. The exophthalmos depends on the amount of effusion and its character. The nerves may be gravely damaged when the general orbital inflammation is slight.

Pl. II. 3 affords an example of the occurrence of simple atrophy of the nerve due to this cause. It is an illustration of a well-marked type,[1] in which loss of sight of one eye comes on simultaneously with paralysis of all the ocular

[1] For example, those recorded by v. Graefe, "Arch. f. Ophth.," vol. i. pt. 1, p. 424, and Baumeister, ibid. vol. xix. pt. 2, p. 264.

muscles, sometimes with tenderness on pressing the eyeball back into the orbit. The symptoms have been ascribed to hæmorrhage (v. Graefe) or inflammatory mischief (Baumeister) at the back of the orbit. In the case illustrated, the cause was almost certainly "rheumatic" inflammatory mischief, for the symptoms came on suddenly, with much pain, after exposure to cold, in an intensely rheumatic woman, who had previously had an attack of "rheumatic" paralysis of the facial nerve. The paralysis of the ocular muscles passed away, but that of the optic nerve persisted, and the disc slowly passed into atrophy without the least sign of neuritis. In such a case it is probable that the nerve suffered chiefly from pressure. A case of rapid but not permanent failure of sight, accompanied with shooting pains passing to the back of the head, in a woman who had had facial paralysis, has been recorded by Nettleship.[1] There was slight puffiness of the eyelids, but no tenderness on pressing the eyeball back, and the ophthalmoscopic appearances were normal.

In another case, probably of syphilitic mischief at the back of the orbit, with intense pain in the eye, orbit, and head, the inflammation descended to the eye, and produced secondary papillitis, ending in atrophy. In this case sight was lost, and the vision of the other eye also became impaired. Hence it is probable that the inflammation extended from one optic nerve to the other, probably by the chiasma—a danger that makes energetic, prompt treatment imperative.

A very similar state of secondary atrophy of the nerve may result from a blow on the head,[2] or on the eye. Rapid exophthalmos and the appearance of the lids may show that hæmorrhage has occurred into the orbit. These conditions are considered in the section on "Injuries to the Head."

In rare instances, hæmorrhage has occurred apart from injury, in sufficient quantity to cause prominence of the eyeball and distension of the eyelids with blood. Of two

[1] "Lancet," 1881, i. p. 760.

[2] Snell: "Ophth. Rev.," i. 402.

cases recorded by Ayres,[1] the exciting cause in one was a violent effort, in the other a strain during vomiting. The degree of impairment of sight appears to depend upon the amount of blood effused, and the consequent stretching of the optic nerve.

Tumours in the Orbit.—A tumour at the back of the orbit or of the optic nerve, may cause neuritis such as results from intra-cranial tumour, but this is at first limited to the eye in front of the growth; the other optic papilla either escapes or presents only a slighter and later inflammation, which has been communicated to the nerve through the chiasma. There is also distinct and increasing prominence of the eyeball.

Injuries to the Head.

Injuries to the head, blows, falls, &c., frequently cause ocular symptoms and often very marked ophthalmoscopic signs. The forms of ocular affection are of several varieties.

1. Impairment or loss of sight, without ophthalmoscopic changes, or with very slight alterations—simple congestion of the disc, easily overlooked. Such impairment may result from blows on the anterior portion of the head. In some cases the mischief is probably direct concussion of the retina, for in slight cases an alteration of vision has been noted such as must be ascribed to disturbance of the retinal elements. For instance, in a case recorded by Gosetti, after a blow on one angle of the orbit, near objects appeared unduly large, and there was some colour-blindness, but no ophthalmoscopic change.

2. Optic neuritis has followed injuries to the head in many cases, at an interval of a few days or weeks. It is apparently due to secondary results of injury, especially to meningitis (Pl. III. 5), less commonly to traumatic inflammatory softening of the brain or hernia cerebri. In a case under my own observation, there was a compound depressed fracture of the left parietal bone. This was elevated five weeks after the

[1] "Archives of Ophthalmology," vol. x. pt. 1, March, 1881, p. 42.

injury, but a few days later hernia cerebri occurred. The optic discs were then normal, but five days later there was acute optic neuritis, which persisted until death.

The neuritis may be slight or considerable, and may entail loss of sight and consecutive atrophy. When occurring long after an injury it may be due to abscess of the brain, as was possibly the case in a patient who presented double papillitis a year after a violent blow from an exploded shell, over one eyebrow, which ultimately caused necrosis of bone.[1]

3. Simple atrophy of the optic nerves, unilateral or bilateral, may result from injuries which damage the optic nerves, directly or by pressure from secondary inflammation. An example of this condition was met with in a patient in whom a fall on the right side of the head and shoulder, injuring the circumflex nerve, was followed by slow grey atrophy of the right optic disc. In such cases sight often fails some time before the ophthalmoscopic signs of atrophy are apparent. See below, "Fracture of the Skull."

4. In some cases an injury to the head may be followed by gradual failure of sight, with very slight and stationary papillitis. In such cases it is probable that a chronic interstitial neuritis has been set up in the nerve trunk.

Concussion of the Brain is attended by no ophthalmoscopic change. Simple concussion of the nerve and retina may probably, as just stated, cause loss of sight and slow atrophy.

Contusion and Laceration of the Brain may entail optic neuritis, commonly slight in degree, although sometimes marked with increased vascularity and redness and opacity of the adjacent retina. It is apparently due, in some cases, to a secondary meningitis, but may occur directly from the brain lesion. It may constitute a valuable indication of the occurrence of greater mischief than a mere concussion. For instance, in a case recorded by Gazet,[2] the symptoms of concussion were followed by neuritis and consecutive atrophy, and ten weeks after the injury the necropsy showed two foci

[1] Recorded by Boncour: "Journ. d'Ophth.," July, 1872.

[2] "L'Union Méd.," 1865, ii. 3, No. 63.

of red softening in the right anterior lobe and one in the corpus callosum. Panas has found in such cases distension of the sheath of the nerve, and it is assumed, on the Schmidt-Manz theory, that thus the neuritis is produced, but this is at present unproved.

Fracture of the Skull[1] not uncommonly causes loss of sight in consequence of laceration of the optic nerve. According to the statistics of Hölder, quoted by Berlin, the orbital vault is involved in 90 per cent. of fractures of the base of the skull (80 out of 88 cases), and the optic canal is implicated in 54 (or 60 per cent.). In 42 of these there was hæmorrhage into the sheath of the optic nerve. The most frequent causes are blows and falls on the frontal bone (especially the orbital portion), less frequently on the temporal or occipital bone. The effect of the resulting laceration of the nerve is usually immediate and permanent loss of sight. It is generally unilateral and on the side of the injury, very rarely on the opposite side, as in a case recorded by Leber and Deutschmann, in which the eye blinded was on the side opposite to that on which blood escaped from the ear. Both eyes are only affected when both optic canals are fractured. Sometimes the hæmorrhage into the orbit is evidenced by prominence of the eyeball and effusion of blood into the eyelids. The optic nerve may be torn, compressed, stretched, or the seat of hæmorrhage. Absolute loss of sight from direct injury to the nerves is usually permanent. When the lesion, as is commonly the case, is behind the place of entrance of the central vessels, there is at first no ophthalmoscopic change or only transient retinal hyperæmia, but atrophy gradually sets in. The pallor has been observed to commence three weeks after the injury. The ultimate appearance of the disc is usually that of simple atrophy, the edges sharp, and the vessels of normal size. Sometimes narrowing of the

[1] The statements in the text are, in part, derived from important papers by Berlin ("Heidelberg Ophth. Gesellsch.," 1879, and "Annales d'Oculistique," vol. lxxxiii., 1880, p. 69), and by Leber and Deutschmann, "Arch. f. Ophth.," vol. xxvii. pt. 274. See also Graefe and Saemisch's "Handbuch," vol. v. p. 219.

vessels has been observed, and has been ascribed to the extension of inflammation to the tissue around the vessels, or to their direct compression by the injury, or by effusion of blood. Ophthalmoscopic signs of inflammation are not common, except as a result of subsequent meningitis, but, in one of my cases, œdema of the disc with retinal hæmorrhages accompanied effusion of blood into the optic sheath. If the injury to the nerve is in front of the place of entrance of the central artery, the ophthalmoscopic appearances are similar to those of embolism. When the injury to the nerve is partial, the loss of sight may be incomplete, and in such cases central scotomata and peripheral limitation of the field have been observed. When sight is impaired by effusion of blood into the sheath, the prognosis is said to be better than when the nerve is injured. Occasionally signs of direct injury to the eye have been observed in these cases, rupture of the choroid or vitreal opacities.

Compression of the Brain may, it is said, be attended by changes in the fundus oculi—distension of the retinal veins, congestion and œdema of the papilla. Such appearances are, however, certainly rare.

Traumatic Meningitis entails, very commonly, ophthalmoscopic changes similar, for the most part, to those which are found in tubercular meningitis. Meningitis often results from fracture of the base of the skull, and may, like tubercular meningitis, be attended with neuritis. An instance of traumatic mischief with neuritis is afforded by the case figured in Pl. III. 5. The neuritis came on with mental disturbance and convulsions, following, at an interval of a week, a fall on the head. The change was slight in degree, although very distinct, and passed away soon after the cerebral symptoms subsided, leaving no trace. When the neuritis is more intense, blindness may result. Hock[1] has described the case of a child who had symptoms of meningitis five months after a fall on the head. Optic neuritis ("descending") was found with the ophthalmoscope, sight

[1] "Oest. Jahrb. für Pädiatrik," vol. v. 1874, p. 1. "Nagel's Jahrb. f. Ophth.," vol. v. p. 427.

being little impaired. Four years later, however, the child was healthy but blind, with atrophy of both optic nerves. In other cases of the kind actual meningitis has been found. The neuritis may be associated with the signs of mischief at the base of the brain, paralysis of ocular muscles, &c. The chronic inflammatory consequences of an injury (chronic meningitis, inflammatory "growths," &c.) may persist and progress for a long time, even for years, as in a case in which meningeal growths, apparently the result of chronic inflammation, were found beneath two old fractures of the skull, the result of injuries received several years previously. At the base the results of chronic meningitis had damaged the arteries and caused fatal softening, but no recent change.

Hernia Cerebri, resulting from fracture of the skull, with loss of bone, may be accompanied with neuritis, as in the case referred to on page 183.

Necrosis of the Cranial Bones.—The damage to bone by injury may cause necrosis and meningitis or abscess of the brain, both of which may entail inflammation of the optic nerve. As Hughlings-Jackson has pointed out, the relation of the symptoms to the injury may be obscure and unsuspected by the patient or friends, so that careful attention should be paid to any sign of injury, such as puffy swelling, &c., and the occurrence of a blow or fall should be carefully inquired for in all cases of local brain disease.

Diseases of the Nose.

Some curious cases have been recorded[1] in which optic neuritis coincided with persistent discharge of watery fluid from one nostril. In most cases there were chronic cerebral symptoms, and in some there were polypoid growths in the nose.

The fluid is not cerebro-spinal fluid, nor is it ordinary nasal

[1] See Nettleship: "Oph. Rev.," 1883, and Emrys Jones: "Oph. Rev.," vii. 97.

secretion. The most probable explanation is that there is increased intra-cranial pressure and hydrocephalus, and that the escape of fluid relieves the pressure indirectly, and that it is conditioned by some abnormal state of the mucous membrane of the nose. In a case recorded by Baxter,[1] however, there was no disease, but the bones of the skull were abnormally thick. But in this case the cerebral symptoms were rather those of functional than of organic disease. To increase the mystery, some of the cases presented slight symptoms of exophthalmic goître.

Insolation and Heatstroke.

The occurrence of congestion of the optic discs in cases of severe sunstroke, described by Macnamara, has been before alluded to (p. 138). In America, according to Hotz,[2] it is not uncommon to meet with cases of atrophy of the optic nerves, which are ascribed by the patients to sunstroke. Commonly the arteries are narrowed, as if from preceding inflammation, and in some recent cases he met with actual neuritis. In three the exposure had been to the sun, in three to an intense heat. Severe headache was a prominent early symptom, and it is probable that the neuritis was secondary to acute cerebral congestion or meningitis.

Hotz has also seen exudative choroiditis apparently from the same cause, in degree sufficient to cause detachment of the retina. He regards it as due to the extension of inflammation along the sheath of the optic nerve, but the absence of choroiditis in other cases of such extension renders the explanation difficult to accept.

[1] "Brain," v. 325.

[2] "American Journal of Medical Science," July, 1879.

DISEASES OF THE SPINAL CORD.

Inflammation.

Spinal meningitis may be accompanied by ophthalmoscopic changes when the cerebral membranes are also affected (see "Cerebro-Spinal Meningitis," p. 178). But we must be prepared to meet with neuritis as a coincidence of any local inflammation that is produced by a blood-state, so many and various are the states of the blood in which the papilla becomes inflamed.

Myelitis is an illustration of the same truth. It is also usually unattended by any changes in the eye, but to this rule several remarkable exceptions have been recorded. They show, conclusively, that the papilla is susceptible to some states of the blood that influence the spinal cord. Slight optic neuritis, veiling the edges of the discs, was seen by Clifford Allbutt in a case of chronic myelitis in the upper dorsal region. Partial grey atrophy supervened. The same observer has also met with partial atrophy after dorsal myelitis. Seguin[1] has twice seen optic neuritis coincident with subacute transverse myelitis. The affection of the optic nerves ran a favourable course and left no impairment of sight. Noyes[2] has recorded the case of a young man in whom, without cause, impairment of sight in the right eye was simultaneous with some spinal symptoms, and a fortnight later slight optic neuritis was found. The spinal symptoms (initial retention of urine, tingling, and some anæsthesia in the legs) did not increase, but the fields of vision became changed in a peculiar and irregular manner, suggesting an affection of the chiasma or optic tracts.

In a man, aged fifty-two, whose case has been recorded by

[1] "Journal of Nervous and Mental Disease," April, 1880.

[2] "Archives of Ophthalmology," vol. ix. 1880, pt. ii. p. 199.

Steffen[1] and by Erb,[2] loss of sight, commencing by a central scotoma, and accompanied by slight neuritis, occurred first in the left eye, and, three weeks later, in the right. Sight slowly returned, but three months later there was renewed failure in both eyes with temporal hemianopia, without marked ophthalmoscopic changes. Two months later the symptoms of a transverse dorsal myelitis came on.

Very significant also are two cases of coincident neuritis and myelitis observed by Dr. Dreschfeld, of Manchester. One was a man aged forty-one, who, simultaneously with an attack of double optic neuritis going on to complete atrophy, and slight mental disturbance, presented the symptoms of acute myelitis, from which he died at the end of a month. The necropsy revealed disseminated acute inflammation of the spinal cord in the dorsal and lumbar regions. The brain appeared healthy. The other case was that of a woman, aged thirty-eight, who died from respiratory paralysis six weeks after the onset of symptoms of acute myelitis. Soon after the paralysis came on, double optic neuritis was found to exist. After death the brain presented merely signs of congestion, but the upper part of the spinal cord was softened.

It is probable that, in these cases, the optic neuritis and myelitis were both the result of a common cause. The coincidence of acute inflammation of the optic nerve and spinal cord is of considerable interest in connection with their frequent affection in chronic disease. It is probable, however, that in some cases (Noyes, Steffen) the cause of the neuritis was situated at or near the chiasma.

Sclerosis of the Cord.

Posterior Sclerosis: Locomotor Ataxy.

Atrophy of the optic nerves is, as is well known, frequent in locomotor ataxy. In what proportion of the cases it

[1] "Sitzungsbericht der Heidelberg Ophth. Gesellschaft," 1879.

[2] "Archiv für Psychiatrie," vol. x. p. 146.

occurs is difficult to say. Ophthalmic surgeons have been impressed with its frequency. Charcot believes that almost all cases of so-called simple atrophy ultimately present spinal symptoms. Careful statistics show that the proportion of the cases of simple atrophy in which spinal symptoms of any kind can be recognized is about one-half (see p. 112).

But we must not infer from this the converse proposition that most cases of ataxy present optic nerve atrophy. It is probably near the truth to say that about one ataxic in six suffers from optic atrophy. Of seventy consecutive cases of ataxy which have come under my observation, only nine presented atrophy. It existed in nine out of fifty-two cases recorded by Voigt,[1] and in seven out of fifty-six cases analyzed by Erb.[2] Thus of 178 cases of ataxy, optic nerve atrophy existed in twenty-five, or 14 per cent. When it does occur, it is more frequently an early than a late symptom, occurring before rather than after the difficulty in walking has become considerable. In the nine cases above referred to, the onset of the atrophy preceded any distinct disturbance of locomotion in eight. In only one case did it develop after the inco-ordination was considerable, and in this the spinal symptoms came on very rapidly. When sight is lost, any inco-ordination which exists is greatly increased—the condition which the physician employs as a test to exaggerate the difficulty, the withdrawal of the guiding visual sensation, being permanent. The ataxic symptoms are often so slight that, even as increased by the blindness, a careful investigation is necessary to discover them. Blind people often walk in a more or less hesitating and uncertain manner, and the uncertainty of slight ataxy is easily attributed to the blindness. Inquiry, however, elicits other symptoms, as pains in the limbs, especially "lightning pains," and loss of sexual power, and careful observation of the gait shows an unsteadiness in turning, and in standing with the feet bare, and toes and heels close together. It is, however, well known that the atrophy may occur before any obvious symptoms

[1] "Berl. Kl. Wochenschrift," 1881, No. 39.
[2] "Deut. Arch. f. Kl. Med.," 1879.

referable to the cord. One extreme instance of this early atrophy has come under my own observation, in which the atrophy of the discs was complete, and vision lost for twenty years before the first symptoms of ataxy showed themselves. I have seen another case in which the loss of sight preceded for sixteen years distinct spinal symptoms. But in many such cases the loss of the "knee-jerk,"[1] an early symptom in ataxy to which Westphal first called attention, precedes other symptoms, and if looked for will often be found to co-exist with optic nerve atrophy when other symptoms of ataxy are absent. A very marked example of this relation, in which the atrophy existed for fifteen years, associated only with lightning pains and loss of the knee-jerk, has been related by Buzzard.[2] Another early symptom is the loss of the reflex action of the pupil to light, although the contraction occurs on an effort at accommodation (Argyll Robertson). The pupils are often small ("spinal myosis"). It is to be remarked, however, that this may co-exist with optic nerve atrophy without any spinal symptom, as in the case mentioned on p. 266.

When the atrophy is advanced, the optic discs are usually grey, even to indirect examination, and to direct examination very grey and mottled, the meshes of the lamina cribrosa may or may not be visible, the edges sharp and clear, the sclerotic ring distinct. Sometimes there is a peculiar gelatinous opacity of the substance of the disc. To ordinary daylight the tint is a greenish grey; to gaslight a bluish or iron grey. Its characters are shown in Pl. II. 6. Less commonly, the discs appear white to the indirect method of examination, but a grey mottling can always be seen with the direct method. The vessels are usually of the normal size. The grey disc and normal vessels have been supposed to be peculiar to this form of atrophy, but this is

[1] It must not be hastily inferred, however, from the occurrence of the jerk, that the atrophy is unconnected with disease of the cord, because lateral sclerosis, in which there is an excess of the knee-jerk, may, in rare cases, be accompanied by optic nerve atrophy.

[2] "Brain," 1878, No. 2, p. 168.

incorrect. The disc in atrophy from post-orbital pressure on the nerve, such as that shown in Pl. II. 3, may present exactly the characters of the atrophy of ataxy.

A stage of hyperæmia, "chronic optic neuritis," has been described by Dr. Clifford Allbutt as sometimes preceding the atrophy, but the occurrence of this condition has not been confirmed by other observers. I have frequently looked for it, but without success.

The anatomical characters of the atrophy have been already described (p. 116). The trunk of the optic nerve is commonly nearly normal in size, but is grey and semi-translucent. The grey degeneration may stop at the chiasma, but often, as Türck pointed out, involves also the optic tracts, and can be traced to the external corpora geniculata. The microscopical investigations (of Leber especially) have shown that the change in the nerve consists of an increase in the interstitial tissue, and sometimes the formation of translucent colloidal tissue around the vessels, as in Fig. 51, p. 117, together with a wasting of the nerve fibres. The histological resemblance to the change in the spinal cord is not so close as has been asserted. Charcot and Abadie have suggested that the change commences in the nerve fibres, and is essentially parenchymatous, but the balance of evidence is not by any means conclusively in favour of this view.

The affection is usually bilateral, although often more advanced in one eye than in the other. In rare cases, one eye may be much affected, and the other very little.

Symptoms.—The affection of sight is usually characterized by a progressive peripheral defect in the field of vision, especially extensive on the outer side (Förster). It progresses until only a small portion is left, situated to the inner side of the blind spot, and enclosing the fixing point. Central vision may be little impaired even after the peripheral defect has become very great. When the acuity of vision is thus preserved patients may, for a long time, be unaware of the affection of sight, until indeed the field is greatly reduced. Sometimes a sector-like defect occurs, an example of which is figured at p. 125. Rarely one half of a field may

be lost (Fig. 56, p. 124). This has hitherto only been observed when the sight of the other eye was entirely lost.

Colour-blindness is frequent, and is almost always an early symptom. The first change is commonly a loss of perception of green, then of red (see p. 120). Occasionally, as I have seen, the defect in the field may be more perceptible in a bright than in a dim light, and the latter be preferred by the patient. The degree of impairment of sight, both in regard to acuity and to colour-vision, may vary from day to day, just as does the degree of impairment of sensibility in the legs.

The manner in which the atrophy often precedes the symptoms of spinal mischief points to the anatomical independence of the two affections, whatever may be their relations. Pathology verifies this conclusion, for in cases in which both posterior columns and optic nerves are affected, no anatomical continuity of degeneration can be traced. The degeneration extends as far as the chiasma, but the tracts are little affected. Thus there is an apparent want of correspondence between the optic and spinal phenomena. Both are, it is true, parts of the sensory nervous system, but in the nerve the seat of the morbid process is peripheral, in the cord it is central. It is, however, asserted by Pierret[1] that this opposition is apparent only. Although the degeneration of the optic nerves can be traced only as far as the chiasma, he has frequently found, in the corpora quadrigemina, anterior and posterior, a process of sclerosis, which thus, at the root of the optic nerve, represents the sclerosis at the roots of the spinal nerves. Further, the latter may, he says, be found changed in the same manner as the optic nerve. On examination of the terminal expansions of the nerves of the anæsthetic and painful regions he has found in two cases the evidence of lesions of these nerves perfectly comparable to that which constitutes optic nerve atrophy. The farther from the peripheral termination the nerves are

[1] Quoted by Robin, op. cit. The statements in the text are partly derived from a communication M. Pierret has kindly made to me on the subject.

examined, the slighter do the changes become, and soon they disappear, and the nerves are healthy, until the posterior columns are reached. Thus, according to this view, in locomotor ataxy we have a combined peripheral and central change in the sensory nervous system; and it has been merely an accident of pathological progress that attention has been primarily fixed on the central alteration in the cord and the peripheral process in the optic nerve. But, as has been pointed out by Gunn, the optic nerve is to be regarded rather as part of the central nervous system than as an ordinary peripheral nerve, and the importance of this relationship must be borne in mind (see p. 112).

The course of the optic nerve atrophy is very like that of the cord degeneration. Recovery of sight, if ever observed, is a still rarer event than recovery of co-ordination in the limbs. The interference with the function of the posterior columns of the cord may, in a recent case, be out of proportion to structural change, but in the eye this is rare, and the structural change is that on which our prognosis is based. At the same time an arrest of progress is sometimes obtained, as it is in the ataxy. Although ultimately almost all cases increase, yet the progress is often very slow, and many years may pass before even a small field is finally lost. The perimeter affords valuable aid in estimating changes, which patients are apt to regard too favourably.

LATERAL SCLEROSIS.

Ophthalmoscopic changes are very rare in cases which present the symptoms of primary lateral sclerosis of the cord. In one or two cases, however, I have seen grey atrophy slowly supervene, similar in character to that met with in locomotor ataxy.

INSULAR (DISSEMINATED) SCLEROSIS.

Amblyopia occasionally occurs in insular sclerosis of the brain or cord, but very rarely goes on to complete loss of

sight. It is often unattended by the ophthalmoscopic signs of atrophy; the examination is frequently difficult on account of the associated nystagmus. In such cases the optic nerves may be found to be occupied by patches of sclerosis, similar to those which occur elsewhere.[1] The nerve fibres passing through are not destroyed, their axis cylinders persist, and retain impaired functional power, although their medullary sheath may disappear.

Occasionally, however, atrophy of the optic nerves is observed in this affection quite similar in its character to that seen in ataxy, attended by a similar loss of vision, progressing to complete blindness.[2] Dr. S. H. Habershon found uniocular central scotoma, absolute for white, red and blue, in a case of this disease. The corresponding optic disc was greyish white, especially in its outer half.[3]

Caries of the Spine.

Caries of the spine in the dorsal region is unattended by ocular changes. Bull[4] has recorded an examination of fifty cases, but the changes he met with, confessedly rare, are of doubtful pathological character, being confined to fulness of the retinal vessels, and sometimes dilatation of the capillaries of the disc. When the caries is in the cervical region, marked congestion of the disc has been described. In one case under my own observation the discs were red, and there was much white tissue about the vessels, very conspicuous against the red disc (as in Pl. I. 2), but the margins of the side were quite clear, and the pathological nature of the appearance was somewhat doubtful. Abadie[5] has recorded a case in which atrophy of the optic nerves supervened, and attributes it to meningitis ascending to the base of the brain, of which,

[1] Charcot: "Leçons sur les Maladies du Système Nerveux," t. i. p. 206.
[2] Magnan: "Arch. de Physiologie," t. ii. p. 765. Liouville: "Mémoires de la Soc. de Biologie," 1868, p. 231.
[3] "Trans. Ophth. Soc.," vol. ix. 1889, p. 162.
[4] "Am. Journal of Med. Science," July, 1875.
[5] "Bull. de la Soc. de Chir.," Jan. 12, 1876.

however, there was no other evidence. In the case of a girl, aged fifteen, suffering from Pott's paraplegia, who was in Queen Square Hospital, under the care of Dr. Buzzard, there was well-marked optic neuritis. She had, however, frequent severe headaches, and occasional vomiting, and the neuritis did not improve as the paraplegia passed away, so that it is possible that some intra-cranial tumour (?tubercular) co-existed.

Injuries to the Spine.

The subject of the changes in the optic discs in spinal injuries has received a large amount of attention in consequence of the prominence which "railway cases" have given to this class of accident. In its scientific relations the subject has not escaped the sinister influence which litigation exercises on the investigation of facts, and there is no doubt that the pathological nature of many of the appearances described in these cases has been the result of an affection of the mind of the observer, rather than of the eye observed. Still, it seems well established that in some cases of spinal injury ocular changes supervene, and the observations of Clifford Allbutt especially show that they occur with greater frequency the higher up the injury is. The changes are those of simple congestion, congestion with œdema, and slight neuritis, uniform redness of the disc, and concealment of the outlines so that the position of the disc may ultimately be recognized only by the convergence of the vessels. In one case a "daffodil colour" was described. Sight is a little, but not much affected, and the condition, which is of slow onset and course (coming on some weeks after the injury), usually passes away. A remarkable case of this kind has been described by Thorowgood.[1] A girl, aged twelve, after a blow on the lower part of the back, complained of pain and tenderness at the neck, with muscular stiffness. A week after this some dimness of sight came on and increased,

[1] "Clin. Trans.," viii. 1875, p. 80.

until five weeks after the blow sight was lost, and well-marked optic neuritis was found. Leeches and mercury were employed, and the discs and sight recovered completely.

It has been supposed (especially by Mr. Wharton Jones) that a disturbance of the sympathetic is the cause of the ocular symptoms in spinal injury. In cases of actual disease of the sympathetic, however, no ophthalmoscopic change has been found (Hughlings-Jackson, Riegel, and Jolly). Clifford Allbutt suggests that they may be the result of "meningeal irritation" passing up to the base of the brain, but other evidence of such irritation has not been recognized.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

Exophthalmic Goître.

The conspicuous ocular symptoms which form part of Graves' disease might lead to the expectation that changes in the fundus oculi would be found in that affection. As a rule, however, it is not so. The prominence of the eyeballs does not lead to any alteration in the optic nerve. The retinal arteries participate in the general arterial dilatation, which occurs so uniformly in the disease, and is ascribed to a paralysis of the sympathetic vaso-motor fibres. The arteries are larger than normal, and when their course is favourable for their comparison with the veins, the two may be observed to be nearly equal in size, clearly in consequence of arterial dilatation. The strong pulsation which occurs in the arteries of the head and neck, in consequence of their dilatation and of the excited action of the heart, may be visible in the retina as a spontaneous arterial pulsation, as Becker first pointed out. He has found it in six out of seven cases, and remarks that it varies in degree, and may at times be unrecognizable.[1]

Chorea.

Embolism of the central artery of the retina is an extremely rare result of the endocarditis which is generally found (post-mortem) to be associated with the disease in

[1] "Kl. Monatsbl. f. Augenh.," Jan. 1880.

severe cases. Only two instances have been recorded; the best marked case is that of Swanzy, of Dublin.[1] The embolism occurred at the time of the commencement of the chorea, and was in the left eye. The chorea was most severe on the left side. The state of the heart is not mentioned. The other case is recorded by Förster, but was not seen until some time after its occurrence. The patient, a child, had suffered from chorea for some years, and during the chorea had lost the sight of one eye. The disc was atrophied, and the arteries very small.

Slight optic neuritis is not very uncommon in chorea, and now and then neuritis of considerable intensity is seen, although seldom in the degree comparable to that usually met with, for instance, in cerebral tumour. I have twice, however, seen this latter intensity attained. In each case the patient was a girl of seventeen or eighteen years, and in each the neuritis passed away completely as the chorea subsided. These cases give significance to the slighter forms. In these the edge of the disc is decidedly blurred, sometimes only on one side, sometimes all round, although not often to such an extent as to prevent its position being recognized in the indirect method of examination. To the direct method the edge of the nasal side is obscured, necessarily. The swelling is slight, the physiological cup seldom encroached upon, and hæmorrhages absent. In all the cases in which I have met with it, there has been a recognizable degree of hypermetropia; this fact would lessen the significance of the neuritis in regard to the chorea, were it not that the aspect of the disc, in every case that I have seen, has become normal when the chorea has subsided. The frequency with which such an appearance is met with is difficult to ascertain; in a percentage probably of eight or ten this is to be seen. The slight form is seen in children chiefly—the more severe in girls about puberty. It is probably, when intense, a co-incident effect of the state of the blood. Slight double optic neuritis was seen by Hughlings-Jackson in a case of hemi-chorea, right-sided. When first observed the discs were

[1] "Ophth. Hosp. Rep.," viii. 181.

hyperæmic, badly margined, the veins large and irregular. The change was most marked in the left eye. The appearance increased with the chorea, and disappeared with recovery. Bouchut has figured white exudation on and about the disc in a case of a third attack of severe chorea. In one case which came under my observation there was also kidney disease, and ulcerative endocarditis, and slight retinal hæmorrhages were also present, so that it is certain that the neuritis could not be ascribed wholly to the cerebral affection.

Neuralgia and Migraine.

Occasionally atrophy of the optic nerve has been observed in cases of severe unilateral neuralgia of the fifth nerve. Its origin is obscure. Temporary amaurosis, supposed to be "reflex," is more common. The transient disturbances of sight, temporary amaurosis, hemianopia, &c., which accompany migraine, are well known. In a girl, aged eighteen, blindness of the left eye occurred after some days of migrainous pain. The ophthalmoscopic appearances were normal; vision was qualitative only, and the pupil did not react to light. Treatment was without effect for fifty days; then chloral and quinine were given, and slight improvement took place, probably not due to the treatment. The slow improvement of vision went on, and the sight became good, although the progress was interrupted by slight relapses due to changes of weather and mental excitement.

Of greatest importance, however, are the attacks of loss of sight lasting for a few hours or a day or two, occasionally observed in the subjects of migraine, apart from attacks of headache, and at other times, in association with pain. This transient failure of sight sometimes remains permanent, always in one eye only. The ophthalmoscopical appearances in such cases are those of occlusion of the central artery. The state is usually ascribed to embolism, but it is more likely thrombosis. Galezowski[1] has recorded three such

[1] "Rec. d'Ophthmal.," Jan. 1882. See also Rampoldi, "Ann. di Ottalmo.," 1882. A case recently described by Doyne was probably of this nature. There had been two attacks of transient blindness of one eye, in the last of

cases without heart disease, also one in which slow atrophy occurred in one eye, and another in which failure of sight after an attack of migraine was attended by signs of neuro-retinitis, with small hæmorrhages and thrombosis in some minute vessels. Now and then atrophy of the optic nerve has been observed to follow repeated attacks, and Hutchinson has associated the three symptoms of migraine, amaurosis, and xanthelasma. Glaucoma is sometimes observed in cases in which there has been long-standing liability to unilateral neuralgia of the fifth nerve. It has been proved that irritation of the fifth nerve may increase the intra-ocular tension.[1]

Idiopathic Epilepsy.

Inter-paroxysmal State.—In idiopathic epilepsy the appearance of the fundus oculi between the paroxysms is, as a rule, normal. Some observers have described changes in the optic discs, and increased vascularity, distended retinal vessels, and the like. I have examined very carefully about a thousand epileptics, and have found that in most cases every character of the fundus was such as is presented by persons not epileptic. Now and then an unduly red disc is to be seen, but not more frequently than in persons not epileptic, and in most cases it is explicable by the ocular conditions—a point too little attended to in medical ophthalmoscopy. The only deviation from the normal state of the fundus which has seemed to me frequent, is an unusual equality in size of the retinal arteries and veins. The latter are not, as a rule, larger than normal, and the arteries appear as if large from a lax state of wall. Spontaneous pulsation in the veins has been described by Kostl and Niemetschek[2] as especially frequent in epileptics: it is certainly not

which the upper half of the retina was found to be œdematous. The ascending arteries ultimately became narrowed on the disc, and the lower part of the field remained defective after several months.—"Trans. Ophth. Soc.," vol. ix. p. 148.

[1] Hippel and Grünhagen: "Arch. f. Ophth.," vols. xiv. and xvi.

[2] "Prager Vierteljahreschr.," vols. cvi. and cvii.

more frequent in them than in individuals who are not epileptic.

During the paroxysm the appearance of the fundus has been described variously by different observers. For obvious reasons, the difficulties in the examination are great, and opportunities are rare. The only change which seems well established, is that the retinal veins, during the stage of lividity, become much distended. Regarding the state of the arteries, there is considerable doubt. On theoretical grounds, because contraction of the cerebral arteries is supposed to be the immediate cause of a fit, it has been expected that contraction of the retinal arteries would also be seen, and De Wecker has described a sensible diminution in the size of the arteries during the pallor, but Kostl and Niemetschek thought that they recognized in one case dilatation of the arteries during an attack. Observation, however, of the size of the vessels by the indirect method, is of small value.

In a case of convulsions from meningeal hæmorrhage, in which there was, however, no initial pallor of face, and also in a case of severe one-sided fits, I have been able to keep a retinal artery and vein under (direct) view through the whole of a severe fit, from before its commencement until after its close. In neither case did the retinal artery present the slightest change in size. During the stage of lividity, the vein became large and dark. In a case of chronic local meningitis of the motor region of the left hemisphere (Case 2), by galvanizing the region of the cervical sympathetic, I was able to produce the aura with which the fits commenced, and once watched the retinal vessels by the direct method during the operation, but no change in their calibre was to be observed, although the aura was so intense as almost to pass into a fit. Clifford Allbutt, during a fit, has observed pallor of the discs, and a similar condition has been seen by Hughlings-Jackson and Arlidge,[1] immediately after a fit, in several cases. During an attack of epileptiform amaurosis, Dr. Jackson failed to see any

[1] "West Riding Asylum Reports," vol. i.

change in the fundus which he was at the time comparing with a drawing of it. After a second attack the veins appeared a little paler than before.[1] I have repeatedly examined patients immediately after fits, but without being able to satisfy myself that there was any difference from the appearance of the disc and vessels at other times. It is possible that, as Knies[2] has suggested, changes in the size of the vessels sometimes described, may be due to a sudden alteration in the intra-ocular pressure from changes in the accommodation.

In cases of epilepsy in which the fits were frequent, Clifford Allbutt has seen hyperæmia of the discs, and even some exudation into them. As a rule, my own observations have given quite negative results. In one case, however, I met with marked changes in the discs, developed under observation during a series of exceedingly severe convulsive attacks, recurring at short intervals for several days. The patient was a young man, and the convulsions were of hysteroid type—paroxysms of struggling, arching of back, throwing about of head and limbs, so intense that the united strength of three or four persons was required to keep the man in bed. They were accompanied by loss of consciousness. Bromide and other remedies produced no effect, and the convulsions continued unabated until ice was applied to the cervical spine, when the attacks at once ceased. The optic discs, after some days of convulsion, became reddened and veiled, so that their edges were quite invisible, and there was distinct swelling. After the cessation of the fits the discs gradually resumed their normal appearance. This patient, about three months later, died, after a series of true epileptiform convulsions beginning in the left hand. Post-mortem, no trace of disease was visible in the brain to naked-eye examination.

It might be expected that the retinal vessels would often give way during the violent venous stasis of an epileptic fit, just as do those of the conjunctiva. As already stated, retinal hæmorrhage is rarely observed under the circumstances, no

[1] "Lancet," Feb. 17, 1874.

[2] "Sitzungsbericht der Heidelberg Ophth. Gesellsch.," 1877, p. 61

doubt on account of the support afforded to the walls of the vessels by the intra-ocular tension.

It must be remembered that many cases of apparently idiopathic epilepsy may present traces of old optic neuritis or choroiditis—indicative, the former certainly, the latter probably, that the convulsions originated in organic brain disease; the choroiditis indicating former syphilis. Traces of old optic neuritis are especially common in cases of epilepsy due to blows on the head. It must also be remembered that chronic convulsions resembling idiopathic epilepsy may occur in the subjects of lead-poisoning and chronic renal disease, in each of which optic papillitis may be present.

Hysteria.

Although functional disturbances of sight (single or double amblyopia, hemianopia, colour-blindness, often with pain on use of the eyes), occur occasionally in the hysterical, ophthalmoscopic changes are very rare. Atrophy of the optic nerve has been met with in one or two cases, but was probably an accidental coincidence; or there may have been co-existent organic disease, such as disseminated cerebro-spinal sclerosis, underlying the manifestations of hysteria. When there is extreme amblyopia, dilatation of vessels and serous transudation into the retina have been seen by Landolt. The chronic perineuritis described by Galezowski in one case must be regarded as altogether exceptional. In hystero-epilepsy also there are, as a rule, no ophthalmoscopic changes, but after extremely severe and repeated fits, slight alteration may be met with, as in the case described in the section on "Epilepsy."

Insanity.

The frequency with which pathological appearances are to be recognized with the ophthalmoscope in cases of insanity has been very variously stated. The discrepancy between

observers is so great, that it seems certain undue weight has been given by some to appearances which are not uncommon in normal conditions. In fact the ophthalmoscopic appearances in the insane seem, for some reason, to be a favourite subject for observers whose experience of normal eyes is insufficient to enable them to estimate the significance of the appearances seen. The observations in which changes were found in a large proportion of the cases examined must therefore be received with considerable reserve. As an instance of the different conclusions which have been reached may be cited the observations of Tebaldi,[1] who found changes in three-fourths of the cases examined; and of Schmidt-Rimpler,[2] who found changes only in thirteen out of 128 cases, and some of the thirteen he considered as doubtful. An even more striking instance of this discrepancy is afforded by two observers of the appearances in general paralysis, one of whom described atrophy as existing in eight out of every nine cases examined, while the other found hyperæmia in about the same proportion.

It must be remembered, in estimating the significance of the considerable changes sometimes found, that the cases of "organic" brain disease, tumour, softening, chronic meningitis, and the like, in which mental disturbance is prominent, occasionally find their way into asylums.

General Paralysis of the Insane.—This disease is more closely allied to some spinal degenerations than to other forms of mental derangement. Unequivocal changes in the eye have been found much more frequently than in any other form of insanity. Loss of sight has been known since the time of Calmeil as an occasional complication; but in a considerable degree it is rare. Billod noted complete blindness in only three out of 400 cases.[3] The loss of sight has been proved to depend on grey atrophy of the optic nerves, similar to that which occurs in spinal

[1] Nagel's "Jahresbericht," 1870, p. 374, from the "Rivista Clinica," 1870.
[2] "Ann. d'Oculist.," vol. lxxiv. 1875, p. 267.
[3] "Ann. Med.-Psychologiques," 1863.

disease. The retinal vessels have been normal in size or narrowed (Magnan). In its slighter degrees, it affects one eye more than the other, and its occurrence may easily be overlooked unless the ophthalmoscope is used. Even in slight degree it is not a very frequent symptom. Galezowski found it in one only of forty cases examined.[1] Boy, of eighty cases very carefully examined, found commencing atrophy, with amblyopia, in four only.[2] Jehn found distinct atrophy in seven cases out of forty-seven: in four double, in three single.[3]

As in locomotor ataxy, it may be an early event, and may even precede the other symptoms of the disease. Magnan has observed the affection of sight to commence two and four years before the other symptoms of general paralysis. In a case recorded by Nettleship, grey-white atrophy of the disc, in a man aged thirty-five, with slight unsteadiness of gait, was followed, nine months after the onset of the amblyopia, by mental symptoms which developed into general paralysis.[4] Mr. Nettleship has informed me that he has since seen three or four similar cases.

It is said by Jehn and Boy that the amblyopia commences with defective colour-vision, just as it may do in locomotor ataxy. As another point of contact between the two diseases, it is of interest to note that Westphal has shown that sclerosis of the posterior or lateral columns of the cord is occasionally found in general paralysis. It has not yet been ascertained whether atrophy of the optic nerves is especially common in such cases.

Magnan[5] has found after death the optic nerves grey in colour and sometimes reduced to a third of their volume, and the chiasma and optic tracts also atrophied. The medullary sheaths of the nerve fibres had disappeared; the walls of the vessels were thickened and covered with nuclei. The changes

1 "L'Union Méd.," vol. xxxi. 1866, p. 404.
2 "Thèse de Paris," 1879.
3 "Allg. Zeit. f. Psych," xxx. 519.
4 "Ophth. Hosp. Rep.," vol. ix. p. 178
5 Quoted by Robin: "Des Troubles Oculaires dans les Maladies de l'Encephale," p. 330, 1880.

were most marked in the circumferential part of the nerve, giving rise to a zone of sclerosis from which thick connective-tissue septa extended into the central part of the nerve, limiting irregular spaces containing degenerated nerve fibres. Magnan found analogous changes in the motor nerves to the eyeball. He regards the process as starting from the walls of the vessels, and as part of a general change in the central nervous system, commencing in the superficial layers.

The atrophy usually begins as such in the simple form, but Magnan and Clifford Allbutt have described an initial stage of hyperæmia—uniform redness of the optic discs, with softened edges. Leber and other observers have failed to find this. Well-marked papillitis was found by Boy in one case, and in another he observed small hæmorrhages along a few of the veins. Neuritis was also seen in one case by Jehn. "Peripapillary œdema," a "brownish circle around the papilla,"[1] was observed in some cases by Magnan and Galezowski. Uhthoff found distinct hyperæmia and opacity of the papilla in a case in which sight had failed in one eye for six weeks only, and was reduced to $\frac{1}{3}$ with concentric limitation of the fields for white and colours. Voisin described an undue tortuosity and dilatation of the retinal arteries, while by Magnan and others a grey or white line along the vessels was frequently observed. Jehn described the arteries as of very small size in some cases. Bouchut has figured aneurisms of the branches of the central artery from two general paralytics. Most of the cases I have examined in various stages of the disease presented perfectly normal conditions. In one case only was there the appearance of simple congestion of the disc.

Mania.—During a paroxysm, Clifford Allbutt in one case found pale discs; in others the discs were hyperæmic. Noyes[2] described hyperæmia in fourteen and anæmia in six out of twenty-six cases. Dr. Savage, formerly of Bethlem

[1] The nature of this appearance is questionable. Œdema usually causes a pale halo around the disc as in Pl. I. 3.

[2] "American Journal of Insanity," 1872.

Hospital, has informed me that he has noted pallor of the discs in some cases, and in others undue fulness of retinal veins, but no other change. Of several cases I have examined, in one only was there a pathological appearance, undue and uniform redness of the discs, with distinctly softened edge.

Melancholia.—Most observers have reported the ophthalmoscopic appearances in melancholia to be normal, and with this my own observations entirely agree. Jehn, however, described hyperæmia in some of forty cases examined, and in two there was actual neuritis, which he supposes to be due to meningitis. Neither in mania nor melancholia has Magnan[1] found any change worthy of note.

Dementia.—In *chronic dementia*, Dr. Clifford Allbutt, classing "worn out lunatics of all sorts" in the category, found changes in twenty-three cases out of thirty-eight—in some atrophy, in others hyperæmia. Noyes found hyperæmia in two-thirds of the cases examined, atrophy in none. Jehn and Klein could find no change in the discs in any cases examined.

In *acute dementia* Clifford Allbutt found no change. "Anæmia of the fundus" with "œdema of the retina around the disc" have been described by Aldridge.[2]

DISEASES OF THE URINARY SYSTEM.

Bright's Disease.

In all forms of renal disease loss of sight from uræmic poisoning may occur.[3] Its characteristics are the sudden onset, completeness, the usual absence of ophthalmoscopic changes, excepting such as may have before existed, the preservation of the reaction of the pupil, and the quick dis-

[1] Quoted by Robin, loc. cit. p. 287.

[2] West Riding Asylum Reports," vol. iii.

[3] The association of transient amaurosis with dropsy after scarlet fever was noted in 1812 by Wells ("Transactions of a Society for the Improvement

appearance of the symptom when the blood-state is relieved by purgation or diaphoresis.

To the almost invariable rule that the ophthalmoscopic appearances are unaffected by uræmia, a few exceptions have been recorded. Thus, in a case of uræmic amaurosis, slight œdema of the papilla, passing away with the return of sight, in the course of a few hours, was observed by Dobrowolsky.[1] Again, Litten[2] has recorded a case of granular kidney in which frequent uræmic attacks occurred with coma, convulsions, and vomiting. Characteristic albuminuric retinitis was present, and a considerable amount of œdema of the papilla, causing swelling and peripapillary cloudiness. During each attack of uræmic symptoms the swelling of the papilla and the adjacent opacity increased, and the veins became more tortuous. After the attack was over, the changes resumed their usual degree.

In diseases of the kidney of considerable duration, the vessels of the retina may present changes which they undergo in common with the vascular system of the body generally. The tendency to hæmorrhage which exists in so marked a degree in many cases of chronic Bright's disease may lead to simple retinal hæmorrhage. Lastly, considerable changes are often seen in the retina, which vary greatly in different cases, and are commonly described by the general, but not very accurate, term of "retinitis albuminurica."

Vessels.—According to my own observations,[3] in some cases of chronic renal disease, especially of the granular form, there is to be seen a notable diminution in size of the retinal arteries, independently of the existence of any special

of Medical and Chirurgical Knowledge," vol. iii.). The first observation of actual changes in the retina was made (post-mortem) by Türck in 1850 ("Zeitschrift der Wiener Aerzte," No. 4, 1850). The microscopical changes were first carefully studied by Zencker ("Arch. für Ophth.," ii. 142) and Virchow ("Arch. fur Path. Anat.," x. 1856, p. 178).

[1] "Klin. Monatsbl. für Augenheilk.," March, 1881, p. 121.

[2] "Charité Annalen," 1879, p. 169.

[3] "British Medical Journal," December 9, 1876.

retinal disease. The veins are in such cases not larger than the normal, but the arteries are not more than one-half or even one-third the diameter of the veins (Pl. IX. 2), instead of being two-thirds or three-quarters the diameter. The comparison can only be made, as already stated (p. 9), between arteries and veins which run side by side and correspond in distribution. Sometimes the arteries can be seen, even by the direct examination, as lines only (Pl. IX. 4.) I have only observed this, however, when papillary obstruction co-existed. The size of the arteries may then be less than is ever seen in simple papillitic obstruction without Bright's disease. Very often, when slight swelling of the retina co-exists, the arteries are invisible beyond the papilla (Pl. IX. 3, X. 1), due in part, I believe, to their extremely small size. When this reduction in size exists the pulse usually presents marked incompressibility. A reduction in size, in one case of acute passing into chronic Bright's disease, was observed to coincide with a very marked increase in the tension of the pulse. The contraction is not visible, however, in all cases in which the arteries are tense. In the absence of any cause for the reduction, it must be ascribed to arteriole contraction, and constitutes evidence of some weight in support of the view of Dr. G. Johnson, that such contraction exists, and causes the hypertrophy of the muscular coat of the artery. It is, as just stated, to be seen, in some cases, independently of any retinal disease, but is not invariable even when the tension of the pulse is very high. This may in some cases be due to degenerative changes in the walls of the vessels, as in Pl. XII. 1, in which no contraction can be perceived.

According to Brailey and Edmunds,[1] the walls of the retinal arteries are constantly altered in chronic Bright's disease, even when no abnormal appearances can be seen with the ophthalmoscope. The thickening consists of a growth of tissue which is especially situated between the endothelium and the rest of the interna. It may progress even to the obliteration of vessels.

[1] "Trans. Ophth. Soc.," vol. i. p. 44.

When the retina is diseased, conspicuous white lines are sometimes seen along its vessels, apparently due to a sclerosis of the outer coat. I am not aware that this condition has been observed in any case in which the retina was otherwise normal. The remarkable appearance shown in Pl. XII. 1 presents, however, a still more extreme condition of perivascular change. The arteries are, in part, concealed by a white opaque sheath, ceasing in places suddenly, and presenting the normal vessel emerging from the sheath.

In the same fundus one artery presented two small aneurismal dilatations—an interesting evidence of the vascular degeneration which is a well-known consequence of chronic renal disease.

In the retinal capillaries irregular dilatations may be found, especially in cases of retinal degeneration, as in Fig. 68, p. 218. In this figure an increase of the nuclei of the capillary wall is seen in places, thickening it. It is probable that the degeneration of such nuclei, and the formation of such aneurismal dilatations, are the conditions which lead to hæmorrhages, which were numerous in this case (Pl. X. 1).

Hæmorrhages form, as will be immediately described, a conspicuous feature of most cases of retinal disease in albuminuria. Their common seat is the nerve-fibre layer, in which they are striated and flame-shaped, and often follow the course of the vessels. Less commonly they may occur in other layers, and are then rounded and irregular. They may detach the retina from the choroid or burst through into the vitreous. They sometimes occur, however, apart from other retinal changes, as isolated evidence of the hæmorrhagic tendency. An instance of this is shown in Pl. IX. 1. The retina which presented this extravasation, even up to the time of the patient's death, several months later, showed no sign of other changes. The hæmorrhages are probably due to the weakening of the wall of the minute vessels (by such changes as have been just described), and to the increased intra-vascular tension, causes which are the same as those

which give rise to the extravasation into the brain, so common in the same cases.

"Albuminuric Retinitis."—The special retinal alterations which occur in renal disease are perhaps the most frequent ocular changes to come under the notice of the physician. They are met with only in chronic forms of renal disease—those which are chronic from the beginning, or which are chronic as resulting from an acute attack. They have been met with in most chronic forms of kidney disease—granular kidneys, large white kidney, sequential to an acute attack, and lardaceous kidney. They are by far the most common in the granular form, and least common in the lardaceous kidney.[1] The tendency to their occurrence is said to bear some relation to the amount of albumen in the urine.

Both eyes are almost invariably affected. Yvert, however, records a case[2] in which recovery took place, where the left eye only was affected. The appearances were quite characteristic, with numerous hæmorrhages. The intensity of the changes varied with the amount of the albumen. A subsequent post-mortem showed that the left kidney only existed, and that that was diseased. Yvert assumes the influence of reflex nervous impressions as well as of blood-states, and quotes several cases in which a blow on one lumbar region caused anasarca, limited to, or greater on, that side. This view receives some support from a case of Eales',[3] the sight of whose left eye failed the day after an injury to his left lumbar region. Three weeks later there were white spots near the macula, along with yellowish exudation round the disc, and slight papillitis. The albumen gradually disappeared, and the eye became perfectly normal.

[1] It has been said that retinal changes do not occur with lardaceous disease of the kidney. Cases have, however, been recorded by Beckmann, Traube, Alexander, Argyll Robertson, and Bull, and one case has come under my own observation.

[2] "Rec. d'Ophthal.," 1883, p. 145.

[3] "Trans. Ophth. Soc.," 1885, p. 126.

The frequency of retinal changes has been variously stated. Published statistics vary between 7 and 33 per cent. Eales,[1] in 100 cases of chronic disease, found retinal changes in 28, or 1 in $3\frac{1}{2}$, and this probably represents approximately the frequency with which they are met with. The variation in the estimated frequency is doubtless due mainly to the relation of retinal changes to the duration of the disease. Only after the kidney disease has been exerting its influence on the system for a considerable time, do these changes occur. They commonly correspond in time with the development of cardiac hypertrophy. This led Traube to assert that the hypertrophy of the heart is the cause of the affection of the retina. But the latter may be found in rare cases, without the former.[2] It is not probable that there is any necessary connection between the retinal and the cardiac change, other than that both indicate a pronounced and prolonged effect of the renal disease upon the system. It is indeed well known that the renal disease is often first ascertained by the discovery of the existence of the ocular change, but this is not opposed to the fact just stated, since the retinal disease is only the earliest discovered symptom in those cases in which the renal affection has been insidious in its onset, and has existed for a long time, and reached an advanced stage, before its symptoms obtrude themselves upon the patient's notice.

It has been suggested that the retinal changes may sometimes precede the onset of the renal affection, but all observed facts concur in showing that the relation above described is the invariable one, that renal disease, usually with more or less albuminuria, precedes the retinal affection. The only cases in which the retinal changes precede the albuminuria are rare examples of granular kidney disease, in which albumen is absent from the urine until a late stage of the renal affection, as in the case of a lady, aged fifty-seven, suffering from hemiplegia, who came to me with perfectly characteristic degenerative albuminuric retinitis in each eye. She had hypertrophy of the heart, with strong aortic second sound

[1] "Birmingham Medical Review," Jan. 1880, p. 34.
[2] Cf. Litten, loc. cit.

and high-tension pulse. Repeated careful examination of the urine, however, failed to reveal a trace of albumen, and the specific gravity was not low. There was a family history of rheumatic (?) gout, and of apoplexy. Two cases are also recorded by Abadie. In one of them there was polyuria.[1]

The retinal changes, as a rule, occur only in cases of organic disease of the kidney. In forms of functional albuminuria they have not been observed, with the exception of some cases recorded by Eales.[2] Of 14 cases of young men between eleven and twenty-eight suffering from what was believed to be temporary functional albuminuria, he found retinal changes in 5, white specks in 4, white patches in 1. This observation affords support, as he points out, to the view that, in ordinary Bright's disease, the retinal changes are due to the morbid state of the blood.

The retinal disease presents certain elements which are variously combined in different cases. These are—(1) diffuse slight opacity and swelling of the retina, due to œdema of its substance; (2) white spots and patches of various size and distribution, due for the most part to degenerative processes; (3) hæmorrhages; (4) inflammation of the intra-ocular end of the optic nerve; (5) the subsidence of inflammatory changes may be attended with signs of atrophy of the retina and nerve.

In most cases one or other of these changes predominates, especially in the early stage of the affection, and, according to the element most conspicuous, four types of disease may be distinguished. These are—the degenerative, the hæmorrhagic, the inflammatory, and the neuritic, according as white spots of degeneration, extravasations of blood, parenchymatous retinal inflammation, or inflammation limited to the optic nerve, predominate. It is, however, to be observed that degeneration and hæmorrhage commonly accompany or succeed the inflammatory changes, and that forms are often seen combining the characters of these varieties. In the typical degenerative and hæmorrhagic forms the signs of inflammation are inconspicuous or subordinate.

[1] "La Union Médicale," 1882, p. 627.

[2] Loc. cit. The nature of the cases is open to some question.

The *degenerative form* (Pl. IX. 2) is the most common. It commences usually without signs of inflammation, by the appearance of small whitish spots on the substance of the retina, sometimes near the optic nerve entrance, sometimes at a distance. They are commonly at first soft-edged and rounded, and as they get larger become irregular. Generally, small very white spots, often punctiform or elongated, make their appearance around the macula lutea, arranged in a radiating manner, although frequently not forming a complete circle. These are sometimes so minute as to be only visible on careful direct examination; sometimes they are large and very conspicuous, and are often arranged irregularly, end to end, so as to form radiating streaks, beyond which dots may be scattered (Fig. 67). Often a less intense and diffuse opacity is visible in tracts here and there. Sometimes the larger spots coalesce into white areas, which may surround the disc.

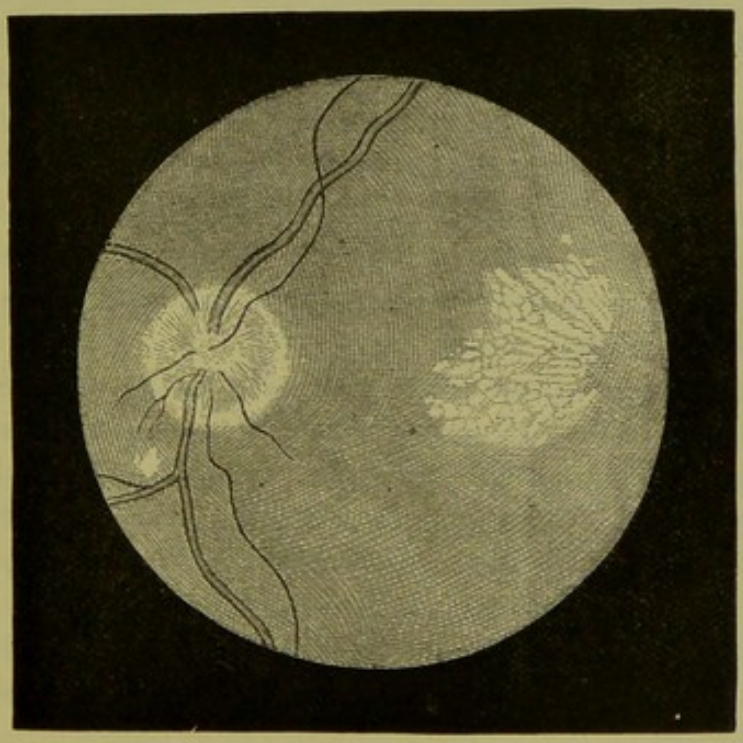


FIG. 67.—THE RETINAL CHANGES IN ALBUMINURIA.

A fan-shaped group of white spots radiating from the macula lutea; small arteries; slight papillitis.

Hæmorrhages, almost constant in all varieties, are slightest in the most chronic degenerative forms. They often are adjacent to the white spots due to the changes in the nerve fibres, and, lying for the most part in the nerve-fibre layer, they have a more or less striated arrangement, determined by the nerve fibres, the direction of which the striæ follow. Sometimes linear hæmorrhages are seen. When larger, the extravasations are more or less flame-shaped. When small, they often lie adjacent and parallel to vessels, but it is not often that the vessel from which they originate can be traced. When large they may be irregular in shape and occupy the deeper layers of the retina.

The diffuse opacity already described is sometimes considerable and accompanied by a little swelling here and there. Such a change is, however, rarely well marked in the form which begins with simple degeneration.

The retinal changes in this form may be considerable without any alteration in the optic disc. Often, however, its edges become blurred, the physiological cup indistinct, and the tint abnormal, reddish-grey.

In two patients suffering from lardaceous degeneration Bull[1] observed the whole retina to present a uniform whitish infiltration, with numerous hæmorrhages. He suggests that the appearance may have been due to lardaceous degeneration of the retina.

In the *hæmorrhagic* form, the conspicuous change is the occurrence of a large number of hæmorrhages, with but little degenerative change and but slight signs of inflammation of disc or retina. Commonly, especially after a time, there is more or less degeneration adjacent to the hæmorrhages, and traces of the halo of spots around the macula are rarely absent. The hæmorrhages, for the most part, resemble those just described, differing only in their number, size, and predominance.

In the *inflammatory* form (Pl. X. 1) there is a general parenchymatous swelling of the retina with complete obscu-

[1] "American Journal of Med. Science," Oct. 1879.

ration of the disc. The vessels are concealed, the arteries especially. The veins are distended, and sometimes have an extremely irregular and tortuous course over the fundus; the arteries are narrow. Hæmorrhages invariably occur in considerable number, and are often large and striated. White spots are commonly numerous, and more or less uniform in character, especially in the acute cases, in which they are large, rounded (as in the figure), and soft-edged. In these cases there is rapid degeneration of the tissue elements, and abundant infiltration with lymphoid cells. If the inflammation subsides, the signs of degeneration may become more predominant, and the optic nerve may present evidence of secondary atrophy. I believe, however, that it is rare for any subsidence of this form to occur, because it is confined to cases in which the effect of the renal disease on the system is intense, and usually soon leads to death.

Neuritic Form (Pl. IX. 2, 3, 4).—In some cases the inflammation of the optic nerve predominates over the other retinal changes to such an extent that it may appear to be the only alteration, and may present nearly the aspect which is common in intra-cranial disease. The edges of the disc are veiled under a greyish-red swelling, of moderate prominence, which may extend a little distance beyond the normal edges of the disc. The prominence may be slight, or such that the veins form conspicuous curves over the sides. The arteries are usually narrow, and often concealed in the swelling; even the veins may be concealed. On direct examination it is generally conspicuously striated. Frequently, on the surface of the swelling, or apparently beneath its surface, there is a conspicuous white reflection in certain spots (Pl. IX. 2), most distinct on oblique illumination. Occasionally on the surface of the swollen papilla may be very minute white dots (just recognizable in Pl. IX. 3).

A careful examination will show, in almost all cases, signs of slight retinal degeneration, sometimes so slight as to require close attention and careful focussing by the direct method to detect them. Sometimes, as in Pl. IX. 2, there

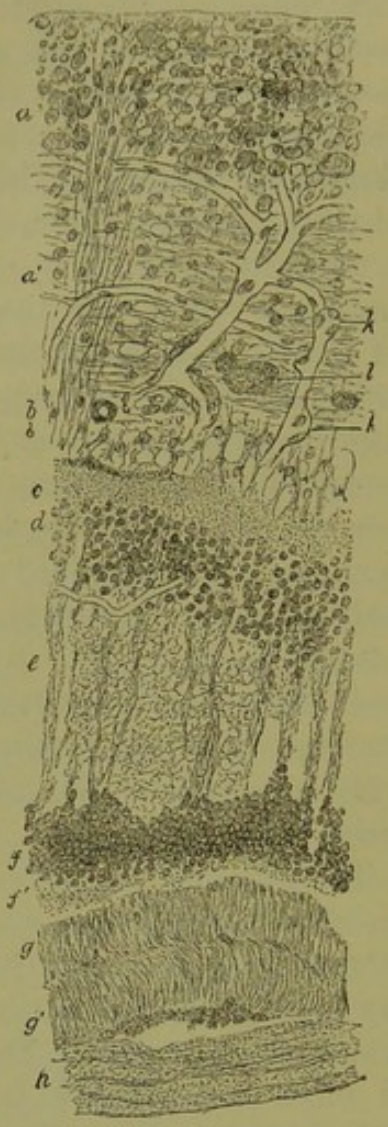


FIG. 68.—SECTION THROUGH RETINA IN A CASE OF ACUTE ALBUMINURIC RETINITIS.

The section passes through one of the white spots near the disc, shown in Pl. X. 1. The retina is greatly thickened, mainly from changes in the nerve-fibre layer (*a a'*), where numerous granular bodies are seen (such as are shown more magnified in Fig. 69). Capillaries are dilated, with conspicuous alterations in their walls; one of them (near right edge of figure) presents a series of aneurismal dilatations (× 180).

are one or two white spots in the retina, near the neuritic swelling. At others, as in Pl. IX. 3, 4, minute white spots are to be detected near the macula lutea. Frequently small hæmorrhages are to be seen somewhere about the fundus (Pl. IX. 4). It is remarkable that there is little tendency

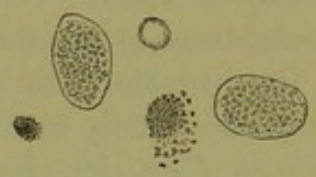


FIG. 69.

PRODUCTS OF DEGENERATION FROM A WHITE PATCH IN A CASE OF ALBUMINURIC RETINITIS. (× 250.)

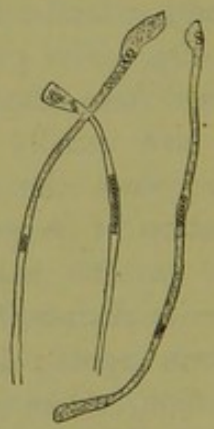


FIG. 70.

DEGENERATED FIBRES OF MÜLLER FROM A CASE OF ALBUMINURIC RETINITIS.

Swelling of the ends of the fibres, and rows of fatty granules due to degeneration. (× 250.)

for hæmorrhages to occur in the swollen papilla in this form. If the neuritis subsides, a condition of consecutive atrophy may be left—a filled-in disc, greyish, with paler lines along the vessels, and often extremely small arteries. Such a condition is shown in Pl. IX. 4.

Anatomical Changes.—The scattered white spots depend commonly on degeneration of the layer of nerve fibres, which are found to be greatly thickened. The fibres often present varicosities, which may attain a large size and become crammed with fat-like globules. These ultimately become isolated as large fat-containing spheres, which, with free globules of fatty matters, are found abundantly on microscopical examination of recent specimens (Figs. 69, 70), and are very conspicuous in a surface view (Fig. 71). The degeneration occurs also, and sometimes chiefly, in the deeper layers, which may also be infiltrated

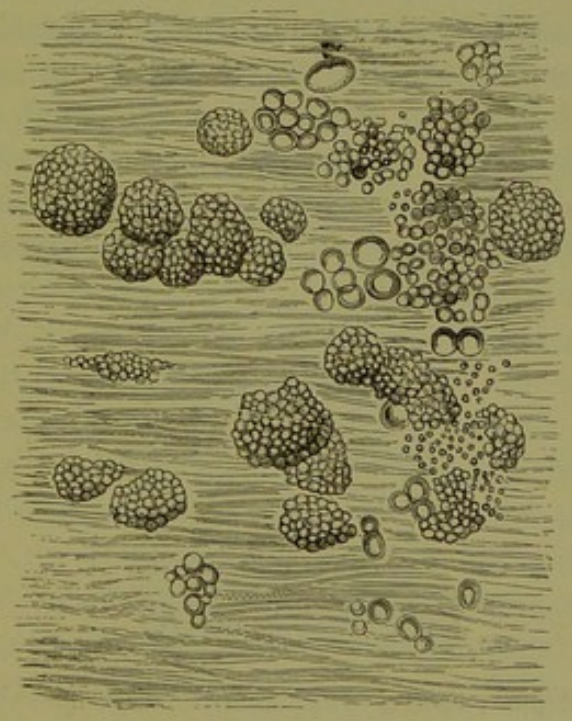


FIG. 71.—SURFACE VIEW OF A WHITE SPOT ON THE RETINA IN ALBUMINURIC RETINITIS.

The transverse lines indicate the nerve fibres. Among these are large and small oil globules and spherules consisting of similar still smaller globules. (After Pagenstecher and Genth.)

with the "compound granule cells." Degeneration of other retinal elements, round corpuscles, and vertical fibres of Müller may sometimes be found. The latter are swollen and contain minute oil globules (Fig. 70). When swollen they have an undue refraction, and have been said, rather unnecessarily, to be "sclerosed." It is to the position of these that the stellate zone of spots around the macula is mainly due. The fibres here have a less vertical direction, radiating from the fovea centralis, and the degeneration of these fibres and the grouping by them of the degeneration of other retinal elements produces the radiating group of spots, most conspicuous near the margin of the fovea, where the fibres become placed more closely together. The diffuse opacity of the retina is in part due to œdema. The elements of the nerve-fibre layer may be separated by clear spaces, and similar spaces may form in the ganglion-cell layer, in the molecular and even in the nuclear layers. In this condition the ganglion cells often fall out of the section (Fig. 72). The diffuse opacity is also partly due to an infiltration of the retinal interspaces with a coagulable fluid, which, after hardening processes, presents an appearance of interlacing fibrillæ with granules at their points of intersection. This may occupy large areas, as in Fig. 68, especially in the

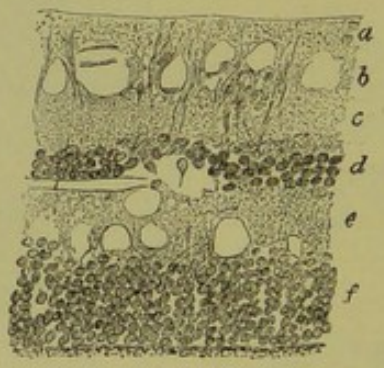


FIG. 72.—SECTION THROUGH THE RETINA, SOME DISTANCE FROM THE DISC, IN A CASE OF ALBUMINURIC RETINITIS, SHOWING ŒDEMA.

The nerve-fibre layer (*a*) is normal, but in the nerve-cell layer (*b*) the ganglion-cells have fallen out, owing to the formation of spaces round them in consequence of the œdema. The other layers show a tendency to dissociation of their constituents, and to the formation of spaces here and there. (× 150.)

outer molecular layer, where cavities, containing this substance and separated by the remains of the vertical fibres, may alone be perceptible. A similar effusion may also separate the "membrana limitans interna" and bases of Müller's fibres from the rest of the nerve-fibre layer. Occasionally the layer of rods and cones presents remarkable thickening, such as is shown in Fig. 68, and is sometimes seen in other morbid states of the retina. Liebreich has called attention to the occurrence of small angular grey spots of pigment, often arranged in groups, and appearing first in the periphery. They are due to changes in the pigment-epithelium, and are seen especially in cases in which a parenchymatous inflammation has passed away.

Choroidal Changes.—Occasionally, although rarely, choroidal hæmorrhage may occur in Bright's disease, and may lead to circumscribed atrophy of the choroid with adjacent pigmentary disturbance. A peculiar "colloid" degeneration of the vessels of the choroid in old cases of albuminuric retinitis has been figured by Poncet. It leads to a thickening of the tissue of the choroid.

Symptoms.—In the slighter forms of the degenerative, hæmorrhagic, and neuritic varieties, vision may be unaffected. More considerable alteration, and even slight parenchymatous inflammation, commonly entails amblyopia, without limitation of the field or changes in colour-vision. In rare cases colour-vision may be affected. As the changes progress, the interference with vision increases. When the macula lutea is damaged, central vision is lost, but this is not common. Degenerative changes rarely reach the centre of the macula, no doubt because the structures in which the degeneration occurs do not extend to the fovea centralis itself. Hæmorrhages, from the paucity of large vessels, are also rare in this situation. The hæmorrhage may, however, encircle the macula, and cause an annular defect in the field. With a central loss of sight, some adjacent colour-blindness was found by Galezowski. Sight is rarely altogether lost. Attacks of uræmic amaurosis often accompany and complicate the amblyopia due to the retinal disease.

Pathology.—We know little of the relation between the renal and the retinal affection. The degenerative changes have been ascribed to the tendency to fatty degeneration which renal disease entails; but this scarcely explains their localization in the retina. Some facts, however, seem to show that a careful recent microscopic examination of the nervous tissues elsewhere may reveal the occurrence of similar changes in them. We know, especially through the researches of Gull and Sutton, that an extensive increase in the supporting tissue of the nerve centres may be found in chronic Bright's disease, and the thickening in the supporting tissue of the retina may be part of this change. Knob-like degenerations of the nerve fibres have also been found elsewhere in the nervous centres.

The facts stated on p. 214 render it probable that the mechanism by which renal disease excites the retinal changes is the altered state of the blood.

The hæmorrhages have been ascribed, with reason, to the double effect of the degeneration in the minute vessels and the increased arterial pressure from the cardiac hypertrophy. It has been speculated that the neuritis may be due to the effusion of serum into the sheath of the optic nerve, but the view rests on no post-mortem evidence.

In several cases in which I have found neuritis predominating, symptoms of cerebral disturbances were conspicuous, intense headache, delirium, convulsions, due apparently to the effects of the blood-state. It seems probable that in these cases there is much cerebral disturbance, and that this may determine the occurrence of the excessive change in the optic nerve.

Complications.—Detachment of the retina is an occasional, although not frequent, accident. It may be double and extensive, as in one case under my own observation. The whole retina was detached in a case recorded by Davidson.[1] It is apparently due to serous effusion between the retina and

[1] "Trans. Ophth. Soc.," vol. i. p. 57; see also vol. viii. p. 141, where Dr. Anderson relates a case in which very extensive retinal detachment occurred in both eyes of a child with chronic interstitial nephritis

choroid. An example of it in slight degree is figured in Fig. 68, which shows that the pigment-epithelium may be detached with the retina.

Hæmorrhage into the vitreous occasionally occurs from the rupture of an extensive extravasation in the superficial layers of the retina. It is always single, and may occur, as in a case under my observation, without the patient's knowledge. One day the fundus was distinct, and vision good; the next nothing but a black reflection from the interior of the eyeball could be seen, and sight was lost. It is hardly necessary to say that damage to vision may be permanent. It may probably occasionally determine glaucoma.

Embolism is said to be an occasional complication of albuminuric retinitis (Voelcker). But this statement must be accepted with considerable reserve. Embolism elsewhere is extremely rare. Thrombosis sometimes occurs in the cerebral arteries, and the signs of embolism may have been due to that cause, and on the other hand the contraction of the retinal arteries may simulate that in embolism; but there is no corresponding defect of the field of vision in these cases, such as would certainly have been present if embolism or thrombosis existed. As I have suggested, the explanation of these appearances which seems most probable is that the tendency to arterial contraction, which is often traceable in normal arteries in this disease, leads to an extreme degree of narrowing when the changes in the disc lessen the flow of blood into the arteries.

Course.—In most cases the retinal changes persist, some lessening, others increasing, until the patient's death. Not rarely, however, they diminish notably, and the retrogression may proceed until the changes almost or quite disappear. This is especially the case when the affection comes on in the course of the chronic kidney disease which results from an acute attack, in which considerable improvement in the renal affection is often obtained, and in other chronic cases when prompt treatment soon after the onset of the retinal disease can improve the action of the kidneys. The effect of purgation in lessening the retinal affections has been often observed, and

Eales has remarked that constipation appears to increase the tendency to their recurrence or relapse. Improvement is often noted in the albuminuria of pregnancy, a form very prone to lead to retinal changes, which commonly improve or even disappear when the pregnancy is over. The greatest improvement is obtained in the cases of slight papillitis. Hæmorrhages constantly disappear, and, if the formation of fresh ones can be prevented, considerable improvement in the retinal state may result. Even the degenerative changes may pass away, especially those which depend on the presence of the granular bodies in the layer of nerve fibres. Most of the white spots shown in Pl. X. 2 disappeared. The most persistent changes are those which result from the degeneration (or sclerosis?) of the fibres of Müller. The white specks around the macula lutea, which result from this cause, rarely disappear. Occasionally remissions in the retinal affection are observed, although the kidney disease progresses. Thus in Litten's case, referred to on p. 209, there was repeated subsidence of the retinal change, in spite of rapid progress of the renal affection. There was not only resorption of extravasation, but also disappearance of white patches.

Diagnosis.—The recognition of the degenerative changes in the retina is only a matter of difficulty when the changes are slight and limited to the region of the macula. The strong contraction of the pupil, when this part is examined, very often renders the use of homatropine indispensable for a thorough exploration.

The aspect of the degenerative form is most closely simulated by the retinal degeneration which results from a neuroretinitis of wide extent (Pl. VIII. 2). It is probable, indeed, that the changes are, to a considerable extent, identical. The damage to and between the radiating fibres around the macula lutea may leave a stellate group of shining spots quite indistinguishable from those which occur in renal disease, and the diffuse white areas nearer the disc may also resemble those seen in the latter form. If the patient have come under observation during the acute period of the inflamma-

tion, there will be no question as to the nature of the retinitic change. It will be seen that, as in Pl. VIII. 1, the neuritic swelling reaches as far as the neighbourhood of the macula, and that the development of the white spots around the latter is part of the changes in the retina occurring near to, and evidently excited by, the inflammation. If, however, the patient come under observation at a later stage, the distinction may be less easy. This is especially the case when a neuritis from a cerebral tumour has been unnoticed till the loss of sight which accompanies its subsidence.

The signs of one or the other classes of disease—encephalic affection or renal disease—are usually, however, sufficiently clear to leave little doubt, after a general survey of the symptoms. But this does not always afford so clear a guide as might be expected. A cerebral tumour may be accompanied by a trace of albumen in the urine. This was the case in a child whom I saw some years ago with the late Dr. Anstie. The only symptoms were headache, the retinal changes, and the trace of albumen. On the other hand there may be no symptoms of intra-cranial disease, except

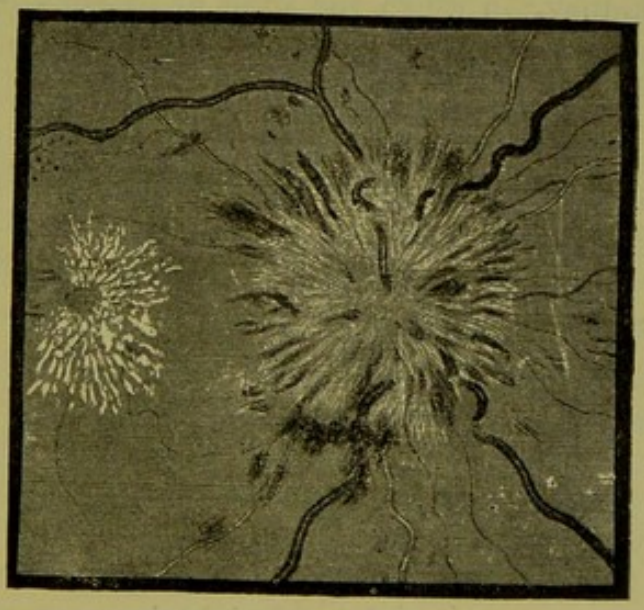


FIG. 73.—PAPILLO-RETINITIS.

From case of cerebral tumour, with appearances at macula closely resembling those common in albuminuric retinitis. (After Edmunds.)[1]

[1] See "Trans. Ophth. Soc.," vol. iv. p. 291 and pl. 7.

headache, which can, alone, hardly be regarded as such, and may accompany the neuritis of albuminuria, as in the case of the patient whose eye is shown in Pl. IX. 3. Lastly, a neuritis, primary in the eye, may occur after diseases, as scarlet fever, which are liable to be attended with albuminuria.

But attention to the following points will, in most cases—I think in all cases—enable a correct diagnosis to be made by the ophthalmoscopic signs alone, or in conjunction with the other symptoms. In the first place, there are always present the signs of a considerable preceding neuritis. Commonly, at the time the failure of sight calls attention to the eye, and the white spots are discovered, there is a prominent pale swelling over the disc, as in Pl. VI. 3. It is very rare for albuminuric neuritis to leave a swelling of this prominence and pallor. If atrophy results from an albuminuric neuritis, the disc, by the time it becomes pale, is very little above the retinal level, as in Pl. IX. 4. Moreover, the neuritic form never occurs, at least as far as recorded facts and my own observation have gone, except in cases of advanced chronic renal disease, commonly of contracting kidney,[1] in which the signs of Bright's disease are always obvious enough. (Regarding these distinctions, see also pp. 96—98.)

In the degenerative changes of neuro-retinitis, of such an extent as to simulate closely the appearance of the albuminuric form, as in Pl. VIII. 2, all the features of the change are those of past, retrogressive mischief. The disc is atrophied, the arteries evidently compressed, and there are, as a rule, no hæmorrhages. In the renal form, of corresponding extent, there are always signs somewhere of active progress. The disc is commonly still inflamed, and there are usually hæmorrhages. Lastly, when the retinal degeneration is present as a consequence of neuritis, at the time any difficulty in diagnosis might arise, sight is almost always lost. Whereas complete loss of sight is an event of great rarity in the albuminuric form.

[1] In one case I have seen it in the late stage of the large pale kidney, in which induration was commencing.

The form in which hæmorrhages and spots of degeneration are combined, may resemble closely the changes in the retina in pernicious anæmia. But in the latter the perimacular circle is commonly not recognizable, and the degeneration is for the most part connected with, and secondary to, the retinal hæmorrhages. The degeneration does not attain the same extent, and the disc is usually unaffected. The same remarks apply, in the main, also to leucocythæmic retinitis. In the latter, the white spots are much more common in the peripheral portions of the retina than they are in the renal form, and in the latter it is very rare to see the circular spots, surrounded by a halo of hæmorrhage, which are so frequent in leucocythæmia. In the latter the tint of the fundus is commonly very different from that in albuminuria. In both pernicious anæmia and leucocythæmia the independent symptoms of the malady usually leave little room for doubt as to the nature of the retinal changes, but it must be remembered that, in the latter especially, renal degeneration is often present.

Prognosis.—Considerable attention has recently been drawn to the unfavourable prognosis as regards life in cases of chronic renal disease with retinal changes. Such patients seldom live two years, and a large percentage of them die within a few months,[1] after the retinal affection is observed.

Treatment.—Local treatment is of doubtful value. Good can only be effected by improvement in the blood-state, especially that which is produced by purgation and diaphoresis. By this means considerable improvement may often be effected in the retinal disease. In several cases, however, the albuminuric spots have entirely disappeared while under observation, although the patient died from the renal affection.

Diabetes.

Diabetes Mellitus.—Defects of sight are common in diabetes (as Bouchardat pointed out many years ago), but changes in the fundus oculi are rare. The most frequent

[1] See Miles Miley, "Trans. Ophth. Soc.," vol. viii. p. 132.

cause for the defect is cataract, which is apt to occur in these cases. Occasionally, considerable amblyopia occurs without ophthalmoscopic changes, probably due to the blood-state and comparable to uræmic amaurosis, although probably the result of a different condition of blood.

Simple atrophy of the optic nerve has been observed in some cases.

In a few cases a central scotoma for white and colours has been observed, peripheral vision being normal. The symptom thus closely resembles that which results from tobacco, but in some of the cases this cause could be with certainty excluded. Examples of this affection have been recorded by Bresgen,[1] Samelsohn,[2] and by Nettleship and Edmunds.[3] The latter, in one of their cases (in which the loss was chiefly for red) found atrophy of nerve-fibres, with increase of nuclei and connective tissue, in a tract which, at the back of the orbit, occupied the axis of the nerve, and near the eye, the outer portion. They attribute the changes in this case, however, to the fact that the patient was a smoker.

Occasionally retinal changes are visible, first observed by Ed. Jäger[4] and afterwards by Desmarres and Galezowski. A careful study of them has been made by Leber,[5] by James Anderson[6] and by Nettleship[7] (Fig. 74). They are only seen when the disease is advanced. In such cases of diabetes, albumen is often present in the urine as well as sugar, but the occurrence of these retinal changes is not related to the albuminuria, since they have been observed in many cases in which not a trace of albumen was present.

The changes in the retina bear considerable resemblance to those of albuminuria, and still greater resemblance to those seen in some cases of pernicious anæmia. Hæmorrhages

1 "Centralbl. für prakt. Augenheilk.," Feb. 1881, p. 33.
2 "Cent. f. prakt. Augenh.," 1882, p. 202.
3 "Trans. Ophthalmological Society," vol. i. p. 124.
4 "Beiträge zur Pathol. des Auges." Wien, 1855, taf. xii.
5 "Arch. f. Ophth.," xxi. 306.
6 "Ophth. Rev.," viii. 1.
7 "Trans. Ophth. Soc.," vi. 331.

are conspicuous in many of the cases, but may be entirely absent, as in the case drawn in Fig. 74. They are often situated behind the vessels, and are sometimes of considerable size. They may exist alone or may lead to a secondary parenchymatous retinitis. In one case, figured by Jäger, a condition of parenchymatous retinitis existed in the posterior segment of the eyeball, with obscuration of the disc, concealment of the veins in places, a few large whitish spots, and a few striated hæmorrhages, the arteries being unconcealed. White spots of degeneration are frequently present, commonly of moderate size, scattered over the fundus. They are situated in the deeper layers of the retina. They differ from the patches of the albuminuric retinitis in *shape*, having less tendency to assume a circular form; in *colour*, having a more dingy shade of white; and in *grouping*, the star round the macula being seldom seen, although there is a tendency for the spots to be arranged in the form of incomplete rings. Sometimes, however, although rarely, there may be a perimacular circle of spots, and this in cases, as those described by Noyes,

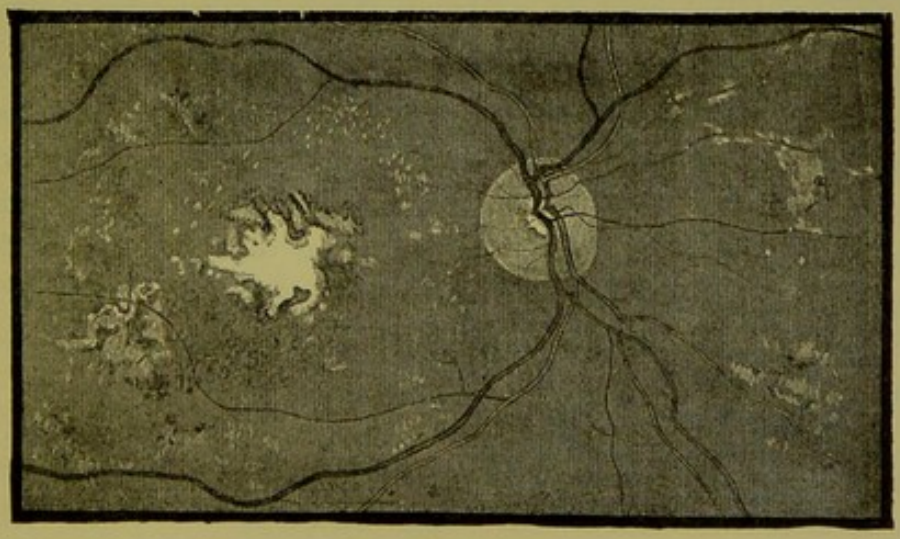


FIG. 74.—OPHTHALMOSCOPIC APPEARANCE IN A CASE OF RETINITIS IN DIABETES. (Nettleship.)

The disc is free from swelling. Scattered about the fundus, especially in yellow spot region, are numerous ill-defined whitish patches (see text). In this case there were no hæmorrhages.

Desmarres, Eales, and Culbertson, in which there is no albumen in the urine. Occasionally a preponderant papillitis may be present, as in the case related by Culbertson,[1] in which consecutive atrophy resulted and caused permanent amblyopia, although the neuritis was apparently cured. The simple atrophy of the optic nerve, which occasionally exists alone, may, in rare cases, accompany the retinal changes (Galezowski).

A marked difference from the forms of retinitis which it most resembles is afforded by the frequent association, in diabetes, of opacities in the vitreous. They appear to be produced by the escape of blood in small quantities from the retinal hæmorrhages. Leber has traced the development of a complete opacity of the vitreous by this mechanism of repeated hæmorrhagic infiltration. Occasionally, hæmorrhagic glaucoma is the result. In one curious case recorded by Nettleship[2] there were, in several parts of the fundus, capillary loops, apparently from the choroid, perforating the retina, and projecting for several millimetres into the vitreous. In another case he found by the ophthalmoscope numerous small dilatations on a large vein near the disc.[3]

Few microscopical examinations have been made. One by Nettleship is recorded by S. Mackenzie.[4] The chief change, beyond œdema, was a peculiar hyaloid degeneration of the interna of the arteries, and numerous capillary aneurisms, some of which are shown in Fig. 75. These vascular changes afford an explanation of the tendency to hæmorrhage. In this case the vessels of the brain (and of the kidneys and spleen) were similarly affected, and a small cerebral hæmorrhage had occurred.

Both eyes are commonly affected in diabetes. The disturbance of sight may be slight or considerable. Blindness is usually the result of the extravasations, or of secondary changes in the vitreous. In Mackenzie's case, just described,

1 "Detroit Lancet," April, 1880.
2 "Trans. Ophth. Soc.," vol. viii. p. 159.
3 "Trans. Ophth. Soc.," vol. viii. p. 161.
4 "Ophth. Hosp. Rep.," ix. p. 150.

the disease was discovered by Waren Tay in consequence of the result of the ophthalmoscopic examination. There is nothing absolutely pathognomonic in the characters of the affection, since they closely resemble the albuminuric form. In addition to the distinctions already described, the most suggestive indications are, as Leber points out, the combination of the retinal change with opacity of the vitreous, and also with atrophy of the optic nerve having the characters of a simple atrophy. In albuminuria, atrophy is very rare, except as the result of neuritis.

The retinal affection is apt to relapse, even though temporary improvement be obtained under the influence of dietetic treatment. The advanced stage of the disease at which it occurs also renders the prognosis unfavourable. The treatment is, in the main, that for the general disease. Carbolic acid is suggested by Leber, but is more likely to be useful in the diabetic amblyopia, without retinal changes, than in the latter.

In very rare cases optic neuritis and glycosuria may both be consequences of an organic cerebral disease. The two

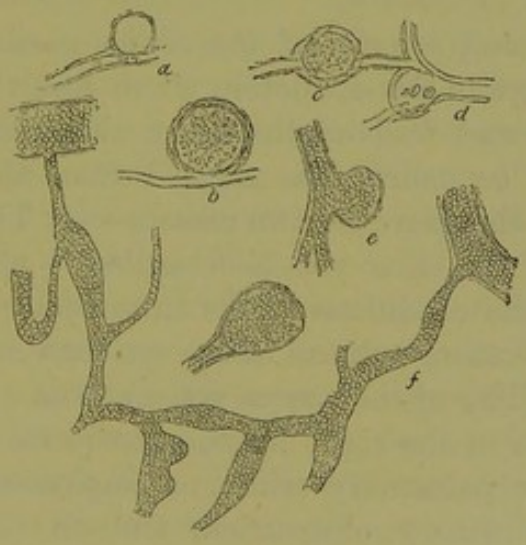


FIG. 75.—CAPILLARY ANEURISMS, AND VARICOSE CAPILLARIES FROM RETINA, IN A CASE OF DIABETES WITH RETINAL HÆMORRHAGES.

They are seen in the course of the vessels (*c*) at their bifurcation (*d*), and also situated laterally (*b*). (× 150.)

symptoms, for instance, existed in a case recorded by Grossmann,[1] and the optic neuritis was thought to be due to the diabetes, until other indications of a cerebral tumour developed. After death a tumour was found in the anterior part of the base of the brain, and the fourth ventricle was distended by a pseudo-membranous mass.

Diabetes Insipidus.—In a very few cases of diabetes insipidus, ophthalmoscopic changes have been observed, which have not, however, much analogy with those observed in diabetes mellitus. Atrophy of one optic nerve was observed by Laycock,[2] and double optic neuritis was present in a case described by Van der Heyden.[3] The connection of these changes is probably with the cause, rather than with the condition, of polyuria. It must also be remembered that the polyuria of contracted kidney is sometimes mistaken for diabetes insipidus.

DISEASES OF THE CIRCULATORY SYSTEM.

Diseases of the Heart.

The veins and arteries of the retina participate in any general changes in the circulation which result from diseases of the valves and walls of the heart, although the changes in them are commonly less marked than those in other vessels. For this there are two reasons—(1) Their size is far below that of the other vessels accessible to physical examination; (2) the conditions of the intra-ocular tension keep the circulation more uniform in the eye than in other parts.

The over-filling of the venous system, from over-distension and dilatation of the right heart, consequent on congenital disease of the pulmonary orifice, on emphysema, and other causes of pulmonary obstruction, and on disease of the mitral orifice, may be revealed by an over-distension of the retinal veins, the chief trunks being large, and the smaller

[1] "Berl. Klin. Wochenschrift," 1879, p. 138.
[2] "Lancet," 1875, ii. 242. [3] "Leyden Thesis," 1875.

veins unduly visible, and therefore apparently more numerous. It is commonly unattended with visual disturbance, although a case in which it was accompanied with transient attacks of amblyopia has been described by Galezowski. This condition is most marked in congenital cyanosis. In that disease the retinal veins may be enormously dilated (as in a case figured in the first edition of Liebreich's Atlas), and they afford proof of the degree to which the distension of the venous radicles contributes to the cyanotic tint. Retinal hæmorrhages occurred shortly before death in a case of congenital cyanosis recorded by Stangloneier.[1] In acute venous over-distension, such as occurs during effort, during severe cough, or during an epileptic fit, the venous congestion may also be very marked.

Under-filling of the arterial system, if chronic, such as occurs in aortic obstruction and in mitral disease, is rarely evidenced by a corresponding state of the retinal vessels, no doubt on account of the second local influence just mentioned.

Nor is chronic over-action of the left ventricle, if sustained, evidenced, as a rule, in the retinal arteries, probably because the cause of such over-action commonly lies between these minute vessels and the heart. Exceptions are, however, met with. In exophthalmic goître, in which the over-action of the heart depends on a primary nervous disturbance, and not on an obstruction to be overcome, distension (and even pulsation) of the arteries may be visible. The former is probably in part due to dilatation of the vessels from vaso-motor paralysis. (See p. 198.)

Sudden over-action of the heart, as from emotion or violent exertion, may also show itself in visible pulsation of the retinal vessels; rarely in the arteries, more frequently in the veins, to which it is transmitted from the arteries.

In aortic regurgitation pulsation in the veins is common, and pulsation in the arteries is not rare. This depends on the fact that the force of the pulse-wave becomes increased out of proportion to the actual movement of the blood, and

[1] "Inaug. Dissert. Wurzburg, 1878; Nägel's "Jahrbuch für Ophth.," 1878, p. 261.

the conditions which obtain in the larger arteries pass on, so to speak, into the smaller vessels, and even overcome the regulating influences of the eye (see p. 20). In one case described, the existence of the valvular lesion was first suspected from this pulsation.

For the above-mentioned reasons, neither simple dilatation nor simple hypertrophy of the left side of the heart usually affects the size of, or circulation within, the retinal vessels. Dilatation only acts when it involves the right side of the heart in an extreme degree, and then may cause some venous congestion. But hypertrophy, when its cause is such as permits it to act on the smaller vessels, may produce, although rarely, retinal hæmorrhages. It is doubtful whether it is capable of doing this unless rupture be permitted by vascular degeneration. The hæmorrhages which result may lead to degenerative white spots, which may persist after the disappearance of the effused blood.

Thrombosis of the central vein occurs in rare cases of heart disease, mitral and aortic (see p. 30).

Embolism of the central artery of the retina is an occasional consequence of valvular disease of the heart, and is probably the most common cause of amaurosis associated with cardiac disease—a coincidence which was first noted by Seidl and Kanka in 1846.[1] Its occurrence is governed by the same conditions as those which determine it elsewhere. It is most common in mitral disease, especially, like cerebral embolism, in mitral constriction. Its signs have been already described (p. 33).

Transient failure of sight without ophthalmoscopic changes is common in heart disease, and may be unilateral and considerable. To the latter form attention has been lately called by Nettleship.[2]

Malignant Endocarditis.—In the rare form of "ulcerative endocarditis" attended with fever and pyæmic symptoms (which Litten better designates "malignant endocarditis"), —the "diphtheritic endocarditis" of some German writers,

[1] Canstatt's "Jahresb.," 1846, iii. 115.
[2] "British Medical Journal," Jan. 14, 1879.

due, probably, to the circulation in the blood of organized elements derived from some septic source—retinal hæmorrhages are almost invariable, and of considerable diagnostic importance. Choroidal[1] and even conjunctival[2] extravasations may in rare cases coexist. Most of the observed instances have occurred after childbirth, and they are described under the head of "Septicæmia" (see Fig. 82, p. 295). Rosenbach,[3] in two cases in which ulcerative endocarditis resulted from experimental damage to the valves of the heart of dogs, found retinal hæmorrhages, minute, in streaks and dots. In these cases, hæmorrhagic infarcts with abundant micrococci, were found in various organs. Hyperæmia of the papilla was associated with the retinal hæmorrhages in a case described by Michel. In the optic nerves were found, after death, many dark points, due to capillary embolism and "miliary abscesses." Extravasations into the kidney were associated with bacterial masses. Virchow has described[4] an interesting case of panophthalmitis (exudation in the iris, vitreous, retina, and choroid) in a case of ulcerative endocarditis in a man, and he found minute bodies in the damaged spots, which would probably be now regarded as micrococci. He also proved by experiment that embolic obstruction of the minute vessels caused punctiform extravasations in the retina. In a case of ulcerative endocarditis recently recorded by J. Hutchinson, Jun., the retina was œdematous, its arteries small, and it showed several hæmorrhages. All these changes may well have been due to the presence of embolism, but other facts in the case rendered a different explanation of the ocular condition possible.[5]

Diseases of the Vessels.

Chronic changes in the vessels rarely reveal themselves by retinal signs. Those which do occur, the rare coincidence of

[1] Westphal: "Arch f. Psychiatrie," vol. ix. pt. 3, p. 389.
[2] Michel: "Arch. f. Ophth.," vol. xxiii. p. 113.
[3] "Arch. für Exp. Path. u. Therapie," 1878.
[4] "Arch. für Path. Anat.," Bd. x. 1856, p. 179.
[5] "Trans. Ophth. Soc.," vol. ix. 1889, p. 152.

aneurisms or signs of degeneration of the retinal vessels, with a similar change elsewhere, have been already sufficiently considered in the general account of the changes in the retinal vessels.

Nor have alterations in the eye been observed in cases of acute lesions of the vessels elsewhere, with the exception of a case of phlegmasia dolens recorded by Walter.[1] The sight of one eye was lost four days after parturition, and a week before the onset of the phlegmasia. Some weeks later, the retina (then first examined) showed extreme contraction of the retinal vessels; the optic disc was pale and the macula reddish. The appearances were supposed to be due to embolism. The retina, however, subsequently became detached.

DISEASES OF THE BLOOD.

Plethora.

In states of plethora it is said by Jäger that the vessels are large, and the blood-column dark. The changes are not, however, sufficiently well marked to be of practical importance.

Anæmia.

Acute Anæmia from Hæmorrhage.—Loss of blood is occasionally followed by affection of vision, and the loss of sight may be slight or complete, transient or permanent, and may come on at the time of the hæmorrhage or not until after several days.

It is remarkable that sight is affected much more frequently after spontaneous than after traumatic hæmorrhage, and in that of the latter form, venesection is the most frequent antecedent, accidental or surgical wounds being very rare causes. This may be related to the circumstance that in traumatic and surgical cases the health is less frequently

[1] "British Medical Journal," April 2, 1881.

impaired before the loss of blood than in the cases in which spontaneous hæmorrhage occurs, or in cases in which venesection is performed. I am not aware that it has ever been noted in cases of the hæmorrhagic diathesis.

For a valuable compilation of the statistics of these cases we are indebted to Fries.[1] Of 96 cases in which the form of the hæmorrhage was noted, in 34 (35 per cent.) the hæmorrhage was from the gastro-intestinal tract; in 24 (25 per cent.) it was from the uterus, in most cases after childbirth, in a few from menorrhagia; 24 (25 per cent.) were due to the artificial abstraction of blood (21 by venesection, 2 by leeching, 1 by cupping); in 7 cases it was due to epistaxis; in 5 to wounds; in 1 case to hæmoptysis; and in 1 to urethral hæmorrhage.

The loss of sight commonly follows a large hæmorrhage, and especially repeated hæmorrhages, but sometimes occurs after a small one. Now and then it follows immediately on the loss of blood (in 26 per cent. of the cases): the patient wakes from the faint to find himself blind. In 19 per cent. it occurs during the first twelve hours after the hæmorrhage. More frequently an interval of two or three or four days occurs between the two events; 33 per cent. occur after the first twelve hours and before the eighteenth day. Fries found that the prompt onset is most common in the cases which occur after venesection, the tardy onset after spontaneous hæmorrhage. Commonly the loss of sight is sudden; rarely, it is preceded by photopsy and pain in the head and back. In one case under my observation neuralgic pain above the eyes occurred after the (post-partum) hæmorrhage, and lasted for several days after the onset of the blindness. In this case each previous confinement had been followed by a similar pain, without affection of sight. Occasionally the same individual has suffered from transient affection of sight after hæmorrhage on more than one occasion, as in a case recorded by Samelsohn, in which temporary blindness occurred after each of several attacks of hæmatemesis.

[1] "Inaug. Dissert.," Tubingen, "Beilageheft zu den Klin. Monatsbl. f. Augenheilk.," 1876.

The blindness is commonly double (in 90 per cent.—Fries), rarely one eye being much more affected than the other. In 10 per cent. of the cases one eye is affected exclusively; in 5 per cent. one eye becomes blind and the other is but slightly affected. The loss is often permanent and complete (in 65 per cent.), the pupils being dilated and not acting to light. Partial or complete recovery takes place in about half the cases (partial, 30 per cent.; complete, 20 per cent.). Leber[1] thinks the loss is commonly more complete when the hæmorrhage is from the stomach, than when from the intestines or uterus, and this agrees with the conclusions of Fries, that improvement, in spontaneous hæmorrhage, is most common after hæmorrhage from the bowels. Complete restoration of sight has been observed after hæmorrhage from the uterus, from the intestines, from the nose, traumatic hæmorrhage, and venesection; never after hæmorrhage from the stomach. Recovery may be much greater in degree in one eye than in the other. It may be complete in both eyes. When the recovery is partial, the field may be limited, but the limitation varies much in different cases. In the case after childbirth, above mentioned, although the sight of both eyes was lost at first, the right recovered with a normal field, while in the left vision was only $\frac{1}{8}$, and the right half of the field was lost. In one case on record, the permanent loss was in the lower half of each field, and was greater in the left than in the right. In another case (Uhthoff[2]), the right field was limited above, and the left field was limited on the temporal side, while the nasal half was lost except in two small islets. The colour-fields were restricted out of proportion to that for white. In a case of Samelsohn's, in one eye central, in the other peripheral, vision was left. A central scotoma was also observed by Mandelstamm.[3] Recurrent transient amaurosis marked one case (Leber).

The ophthalmoscopic appearances some time after the

[1] In Graefe u. Saemisch's "Handbuch," vol. v.
[2] "Arch. f. Ophth.," vol. xxvi. pt. 1, p. 274.
[3] "Centralbl. f. prakt. Augenh." 1879, p. 175

onset, have, in rare cases, been normal. In most cases the disc is atrophied with small vessels, as in the case I have mentioned, in which the disc was greyish-white, the arteries much narrowed, the veins small also, and much new tissue about the vessels in the disc. The degree of narrowing of the vessels, and the time at which pallor appears, have varied in different cases.

In the few ophthalmoscopic examinations which have been made early in the history of the cases, there have commonly been signs of inflammation, usually slight, sometimes intense. The slight changes consist in diffuse opacity of the retina with some œdema of the disc, the more intense in a neuro-retinitis with hæmorrhages. The larger the number of early observations the more does it appear that the permanent damage to sight is related in degree to the intensity of the inflammation.

In one case, recorded by Hirschberg,[1] three days after the hæmorrhage there was slight opacity of the left papilla; distinct neuritis in the other eye without swelling; sight nearly normal. Five days later—R., intense neuro-retinitis, V. $\frac{1}{30}$; L., commencing neuritis, V. $\frac{1}{6}$. The sight of the right eye was lost next day. Three weeks later—R. disc as in neuritic atrophy, V. 0; L. disc merely reddish and indistinct, V. $\frac{1}{4}$. Three years later the patient died of cancer of the stomach: the fibres of the optic nerve were found to be replaced by nucleated connective tissue—there was no evidence of hæmorrhage into the nerve sheath. A very similar case has been recorded by Landesberg. The day after a hæmorrhage from the nose, dimness of sight of one eye was complained of, and the ophthalmoscope showed, in both eyes, diffuse opacity of the retina with some swelling of the papilla. In one eye the appearances soon lessened, and sight was normal; in the other a neuro-retinitis with hæmorrhages developed, with ultimate amaurosis. Retinal hæmorrhages and neuro-retinitis were observed by Woinow after the application of four leeches to the uterus. In a case published

[1] Hirschberg: "Kl. Monatsbl. f. Augenheilk.," 1877. Supplement, 53-85.

by Ulrich,[1] the changes were noted a few minutes after an attack of hæmatemesis. The optic discs were pale, and the vessels on its surface presented a normal appearance. At its edge, however, the veins suddenly lost their dark red colour, and became bright red, like arteries. There were numerous hæmorrhages and white spots along the course of the vessels. This condition of the veins gradually disappeared, and in two months the fundi and vision were normal. The same observer has more recently recorded three additional cases with similar ophthalmoscopic appearances.[2]

Förster has recorded a case, in which, twelve days after a hæmorrhage, there was a peculiar white opacity of the retina, with small hæmorrhages around the disc; vessels small, but not as in embolism. There was no affection of sight. The opacity slowly disappeared without neuritis. So in a case seen by Horstmann, three days after a hæmatemesis, disturbance of sight occurred ($\frac{1}{2}$) with slight opacity of the optic nerves and adjacent retina. The changes gradually lessened, and sight became normal. On the other hand, ten days after loss of sight, which occurred seven days after an abortion, Herter[3] found neuro-retinitis with hæmorrhages, quickly subsiding to atrophy; loss of sight permanent in both eyes. Colsmann, however, four days after onset, found only pallor of disc, small arteries, large veins, and no neuritis.

Many theories have been framed to account for the phenomena, but the variety in the changes renders the appearances very difficult to explain. The theory which has obtained most acceptance is that of v. Graefe, that there is a retro-ocular hæmorrhage situated sometimes near, sometimes far from the eye. The evidence in favour of this theory is that small retinal hæmorrhages have been seen, that there are sometimes hæmorrhages into other organs in cases of loss of blood, and that in one case there were simultaneous symptoms of a cerebral lesion. But this affords a very

1 "Klin. Monatsbl. f. Augenh.," 1883, p. 183.
2 "Graefe's Arch. f. Ophth.," 1887, p. 1. See "Ophth. Rev.," vol. vii. p. 16.
3 "Charité Annalen," 1887, p. 525.

inadequate explanation for the cases in which both eyes suffer. Moreover in those cases which have been examined post-mortem there has been no evidence of such hæmorrhage. To assume, as has been done, that the mischief is at the chiasma, seems unjustifiable, in the entire absence, in all such cases, of other symptoms of mischief at the base of the brain. Förster attributed the slight changes in his case to serous effusion. Horstmann[1] ascribes the symptoms to inflammation in the optic nerves. Ulrich regards papillo-retinitis found after great loss of blood as due to disturbances in circulation in the papilla, and attributes these latter changes to the establishment of an abnormal relation between the blood and the vitreous-pressures.[2] The appearances noted by him in the veins immediately after severe hæmorrhage (see p. 240) he adduces as a proof of these circulatory disturbances. He believes that there is always a slight hindrance to the circulation in the retinal veins where the latter bend in passing over the edge of the disc, that the influence of the intra-ocular pressure is to increase this hindrance, and that it is still further increased by the occurrence of any reduction in the blood-pressure. Severe loss of blood, then, produces a condition favourable to venous stagnation by reducing the blood-pressure markedly, while the intra-ocular pressure is not affected or only slightly diminished. Samelsohn thinks that the nervous connection between the stomach and the corpora quadrigemina (lesions of which are said to cause gastric hæmorrhage) affords the best explanation, while von Oettingen[3] believes that he has proved that fatty degeneration of the retinal vessels, quickly following the loss of blood, is the cause of the extravasations sometimes seen.

It is evident, however, that in the majority of cases there are the signs of inflammation, and there is at present no evidence to show that this is not of intra-ocular origin. It seems probable that the mechanism may vary in different

[1] "Kl. Monatsbl.," 1878, p. 147.
[2] "Graefe's Archiv," xxvi. 3, p. 80.
[3] "Dorpat Med. Zeitschrift," 1877, Nos. 3 and 4, and Nagel's "Jahresbericht," 1877, p. 239.

cases, and one effect of loss of blood may be upon the retinal elements themselves. The shock to the nervous structures from the anæmia may, in some cases, cause transient loss of function, of sudden or slow onset, and recovery takes place without ophthalmoscopic changes. In other cases no recovery may take place, and atrophy supervenes. In some cases the damage to the nutrition of the retina may lead to a primary inflammation on the restoration of the blood-supply, variable in degree, sometimes slight and general, sometimes most intense in the papilla, where inflammation occasionally occurs in chlorosis.

It is probable that more light will be thrown on the pathology of this mysterious accident when physicians are more generally aware of the ocular symptoms which may accompany hæmorrhage, and use the ophthalmoscope in all cases in which the phenomena are likely to occur, since only too many of such cases afford opportunity for post-mortem investigation.

Simple Chronic Anæmia.—Chlorosis.

The colour of the fundus is pale in proportion to the anæmia, but the physiological variations in the tint of the choroid and of the disc prevent the tint of the fundus from affording any absolute indication. In extreme cases the choroidal pallor may, however, be striking, as it was in a girl with chlorosis, lately under my care, in whom the red corpuscles were only 26 per cent. of the normal.

The retinal vessels often present distinct characters. The veins are especially pale, often only a little darker than the arteries. When the choroidal pigment is abundant it may be noted that its influence on the apparent tint of the veins is greater than in health; they undergo a greater change of tint in passing from the dark choroid on to the pale disc. The veins are also often broad, probably in consequence of the defective distension and consequent flattening in their atonic state by the intra-ocular pressure (see p. 10). The reflection from them varies, commonly being broad, no doubt

in consequence of the diminished convexity. The arteries are usually narrower than normal, not merely in comparison with the veins, but absolutely; their pallor is much less noticeable than is that of the veins. The reflection from them may also be broad. Spontaneous pulsation in the retinal arteries has been occasionally observed by Becker[1] in chlorotic girls. Schmall[2] found arterial pulsation in 20 out of 55 cases of chlorosis, the pulsation being usually in the form of locomotion seen at the bends of the vessels. Hæmorrhages are said to occur, but are certainly very rare in simple anæmia, and probably only take place where there is a great absolute deficiency in the number of red corpuscles. I have, however, found them absent in a case in which the corpuscles were only 26 per cent. of the normal.

A valuable paper on the changes found in the fundus oculi in anæmia, based on the examination of fifty cases, has been published by Saundby and Eales.[3] They did not observe arterial pulsation in any of the cases, and venous pulsation not more frequently than in healthy individuals. In five of the cases (10 per cent.) there was slight blurring of the disc, mostly with hypermetropia, and in four there were whitish or yellowish patches of exudation near the disc, or scattered at the periphery. In one case there were two hæmorrhages near the disc; and in several there were small white spots, or small spots of pigment, presumably left by hæmorrhages.

Neuritis occasionally occurs in chlorotic girls. Two undoubted instances have been recorded by me,[4] and one of these is figured at Pl. VII. 5. In each case the anæmia was very great, the hæmoglobin being reduced, out of proportion to the corpuscles, in one to 30, and in the other to 38 per cent. The first case suffered from a relapse of neuritis on a recurrence of anæmia. In each case the improvement was most rapid under the influence of iron.

[1] "Klin. Monatsbl.," Jan. 1880, p. 1.
[2] "Graefe's Archiv," xxxiv. i., p. 37; "Ophth. Rev.," 1888, p. 268.
[3] "Ophth. Rev.," i. 303.
[4] "Brit. Med. Journ.," 1881, i. 793.

The degree of neuritis was slight in the first case, but very considerable in the second. In the case figured in Pl. VIII. 1 and 2, the neuritis must, I think, be ascribed to the same cause. The patient was watched for two years after the subsidence of the neuritis, and, except for an occasional headache, there was never the slightest other symptom to suggest intra-cranial disease. I have never known neuritis from cerebral disease to develope with the extreme rapidity exhibited by this case; and such intensity of progress, in conjunction with the entire absence of cerebral symptoms, excludes, I think, an intra-cranial cause. Iodide of potassium was first given, but no improvement occurred until iron was substituted, too late, unfortunately, to prevent partial atrophy. I fear that the permanent damage to sight was in part due to the delay in the administration of iron. Another case of intense neuro-retinitis with hæmorrhages in a chlorotic girl is recorded by Mr. R. Williams.[1] Rapid recovery took place on the administration of iron.

It is worthy of note that all the patients presented a slight degree of hypermetropia. It is generally admitted that this condition is capable of causing slight congestion of the disc, and if so, it is possible that, in these cases of chlorotic neuritis, the hypermetropia may help in setting up the changes in the papilla which, in the special blood-state, progress to a much more intense degree than they would otherwise attain.

Hirschberg first noted the occurrence of optic neuritis in chlorosis, and an instance, in a girl of sixteen, quickly cured by the administration of iron, has been recorded by Bitsch.[2]

Progressive Pernicious Anæmia.

In pernicious anæmia the tint of the fundus and the appearance of the arteries and veins are such as are seen in the most intense cases of simple anæmia. The rather

[1] "Brit. Med. Journ.," 1884, i. 10.
[2] "Klin. Monatsbl.," April, 1879, p. 144.

narrow arteries and broad pale veins are seen in Pl. XI. 1, from a case which has been published by Stephen Mackenzie.[1] The figure shows also that which is a characteristic feature in pernicious anæmia, the tendency to hæmorrhage. Common in other situations, it is far more frequent in the retina than elsewhere. Of sixteen cases examined by Quincke, retinal hæmorrhages were absent in one only. In thirty cases examined by Horner, extravasations were present "almost without exception." The extravasations are often, as in the figure, numerous, and more or less striated or flame-shaped, from their situation in the layer of nerve-fibres. They are usually most abundant around the optic nerve entrance. They are frequently associated with white spots and areas, due in part to leucocyte-like cells, in part to degeneration in the disturbed retinal tissues, varicose enlargement of the nerve-fibres, giving rise to finely granular, spherical, and fusiform bodies. Homogeneous ("colloid") and finely granular masses have also been found in the inter-granule layer.[2] Occasionally a pale spot may occupy the centre of a small hæmorrhage. In such a case Manz[3] found the pale centre to consist of round colourless cells, sometimes enclosed in a capsule. He found also ampulliform and sacculated dilatations of the capillaries (no doubt similar to those seen in Fig. 75, from a case of diabetes). Some of these were empty, others contained a granular material, others contained red blood corpuscles or colourless cells. He supposes that the capsule found to enclose the pale cells within the hæmorrhages was really the wall of such a capillary dilatation. A stellate arrangement of white specks around the macula lutea was seen by Quincke in one case, but is certainly rare in this disease. Œdema of the retina was also observed in one case by Quincke, the vessels being dimmed by a bluish-white cloud. The optic disc is usually normal, but its edges may be blurred, and optic neuritis may, in rare cases, be present in excess

[1] "Lancet," Dec. 7, 1878.
[2] Uhthoff: "Klin. Monatsbl.," Dec. 1880.
[3] "Centralbl. für d. Med. Wiss.," 1875, p. 675.

of the other retinal changes, as in the case of a boy described by Stephen Mackenzie.[1]

The hæmorrhages are in many cases quickly absorbed, lasting only a few weeks. They cause no disturbance of vision except when located in or near the macula lutea. It is probable that when the actual diminution of the blood corpuscles is ascertained in these cases, a relation may be traced between a certain degree of diminution and the occurrence of these hæmorrhages. In one case the hæmorrhages appeared when the corpuscles fell to 27 per cent. of the normal, and increased with the progressive fall of the corpuscles, which before death were only 12 per cent., and the hæmoglobin 8 or 9 per cent. of the normal.[2]

Scorbutic Anæmia.—A form of anæmia which may be thus distinguished, appears to be a distinct variety of pernicious anæmia. It is characterized by the same progressive pallor and systemic effects of the deficiency of blood corpuscles, but differs in the occurrence of an affection of the gums resembling that met with in scurvy, and in extravasations into the skin. There may also be other cutaneous rashes, such as are met with in cachectic conditions. Hæmorrhages may occur into the retina just as in the ordinary form of pernicious anæmia. It appears not to be in any degree due to deficiency in vegetable food, but to be occasionally produced by abstinence from meat.

An example of this form was described to the Ophthalmological Society by Dr. Stephen Mackenzie.[3] The patient was a lad, aged eighteen, who had had syphilis, but no cause for the blood-disease could be traced. The symptoms were swelling of the gums, enlargement of the glands beneath the jaw, petechial hæmorrhages in the skin, hæmorrhage from the gums, and profound anæmia. Vomiting preceded death. In the retina were abundant fusiform hæmorrhages, gradually increasing in number until there were twenty or thirty in each retina, some as large as the papilla. Ultimately general

[1] "Lancet," Dec. 7, 1878.
[2] S. Mackenzie: "Trans. Ophth. Soc.," vol. i. p. 48.
[3] Ibid., p. 51.

retinal œdema occurred. The corpuscular richness of the blood gradually decreased, during two months the patient was under observation, from 51 to 13 per cent. of the normal. The coloured corpuscles varied in size, some presenting fissures or cracks. The colourless corpuscles were not in excess, except to a slight degree towards the close. They were small and spherical. The hæmoglobin was reduced out of proportion to the corpuscles. No treatment, dietetic or medicinal, appeared to influence the course of the disease. After death, hæmorrhages were found in the lungs and on the surface of the heart.

In a case of my own, the symptoms were very similar to those of Dr. Mackenzie's case. There were the same progressive anæmia, swelling of the gums, cutaneous and retinal extravasations, and hæmorrhages found after death in the lungs and heart. In this case, however, the patient had for a long time abstained almost entirely from animal food, taking plenty of vegetables. There were also, in addition to and accompanying the extravasations, papules with infiltration of the adjacent skin. After death a peculiar change was found in the periosteum of some of the bones.

I lately saw, with Mr. Grellet, of Hitchin, a woman, aged thirty-seven, who presented very similar symptoms, except that there were no retinal hæmorrhages. The affection in this case also came on after entire abstinence from animal food for several weeks, vegetable food being freely taken. There were extreme anæmia, swollen spongy gums, cutaneous petechiæ, and small erythematous spots on the limbs, with a papule in the centre, which became vesicular and formed a scab. Under large doses of iron she had begun to improve, and Mr. Grellet has since informed me that the symptoms passed entirely away.

Leucocythæmia.

In all cases of leucocythæmia in which the change in the blood is considerable, the retinal and choroidal vessels are remarkably pale. The tint of the choroid is usually an

orange-yellow, but if there be much choroidal pigment the tint may be little changed.[1] The retinal veins appear broad and very pale. This apparent increase in width is sometimes very great (Fig. 76), and is probably due to atony and flattening rather than to passive distension. They are often very tortuous. Their central reflection may at first be broad and indistinct; ultimately, in the large tortuous vessels, a very narrow, almost white, reflection appears. The retinal arteries are orange rather than red, and in extreme cases they are small.

Besides these appearances, there are, in a considerable number of cases, actual changes in the retina. These vary greatly in different cases, and rarely present the appearance described by their discoverer, Liebreich, as "leukæmic retinitis." They are almost confined to the splenic variety, and are usually double, one eye being often more affected than the other.

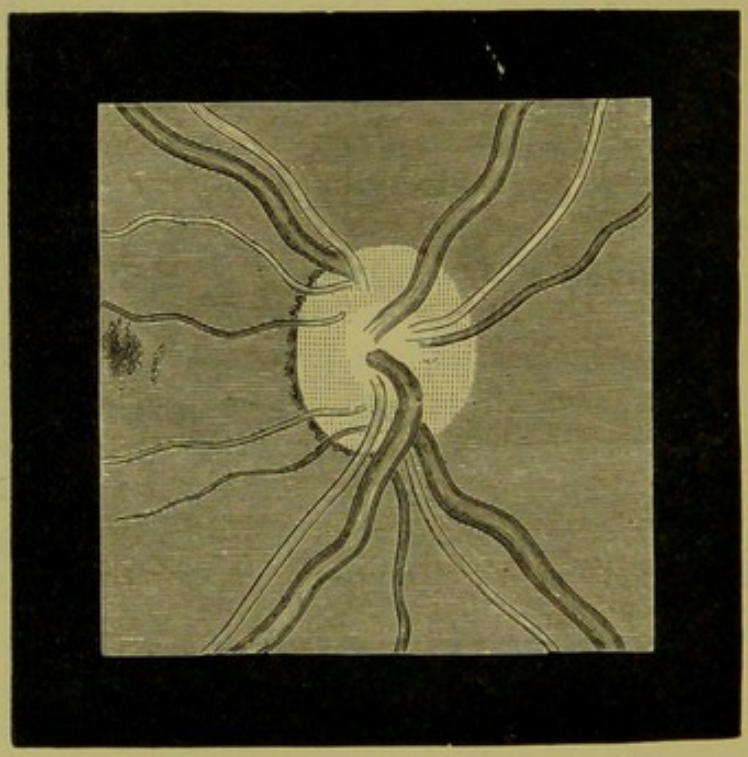


Fig. 76.—Broad Retinal Veins and Narrow Arteries.
From a case of leucocythæmia.

[1] Leber: "Graefe and Saemisch's Handbuch," vol. v. p. 600.

The commonest change is the occurrence of retinal hæmorrhages. The tendency to hæmorrhage in this disease is extremely strong, and leads to extravasations into the retina in a large number of cases. Statistics which I have collected[1] show that the most common recorded seat of extravasation into tissues is the subcutaneous cellular tissue. A more constant use of the ophthalmoscope will probably show that retinal hæmorrhage is as frequent, if not more so. Of five cases of leucocythæmia which I have examined with the ophthalmoscope, retinal extravasations were visible at some period in four. Of the tendency to hæmorrhage these extravasations constitute a striking indication. The tendency to retinal hæmorrhage is apparently far greater in leucocythæmia than in simple anæmia, for it occurs with a percentage of red corpuscles greater than is usual in cases of simple anæmia which present retinal hæmorrhages. I have twice met with them in leucocythæmia when the blood contained 50 per cent. of red corpuscles. One of these cases is figured in Pl. XI. Fig. 2. The hæmorrhage encircles the fovea centralis in a curious series of extravasations, and several smaller ones lie adjacent. Commonly the hæmorrhages are more widely scattered, and more or less striated. The hæmorrhages are in these cases usually in the nerve-fibre layer, but a large extravasation may infiltrate the whole thickness of the retina. When the excess of white corpuscles is considerable, the effused blood has a pale, chocolate tint here as in other situations. Extravasation may take place into the substance of the papilla, or hæmorrhage may occur into the vitreous.[2]

Besides the hæmorrhages, white or yellowish spots are commonly present, often most abundant in the periphery, or near the macula lutea. These are sometimes irregular, but often rounded, and edged by a halo of extravasation. When large, they are sometimes distinctly prominent, and may be as much as 2 mm. in diameter (Reincke). They consist commonly of leucocytes, similar to the leucocytes of the

[1] Art. Leucocythæmia, "Reynolds' System of Medicine," vol. v. p. 257.
[2] *Vide* Perrin and Poncet's "Atlas," pl. 65.

blood, and they have been regarded as lymphoid growths such as occur in other organs. In rare cases, actual growths of some size have been met with, but it is doubtful whether the smaller spots are of this nature. It is common for the pale corpuscles to be aggregated in the middle of an extravasation. In some cases the white spots are due to degeneration of the retinal elements. The capillaries are full of white corpuscles, and it seems more probable that these spots arise by the escape of the corpuscles by diapedesis or by rupture. In one case Saemisch found an irregular thickening of the inner granule layer, in some places extending into the ganglion-cell layer. He attributes the thickening to escaped leucocytes, which are indistinguishable from the corpuscles of the granule layer. Poncet has found a similar infiltration extending, not only into all the layers of the retina, but also into the substance of the optic nerve. Swelling of the nerve-fibres was the cause of small white spots in a case described by Deutschmann.[1] The capillaries of the retina may be dilated and varicose, such as are shown (from a case of diabetes) in Fig. 75.[2] The lymphatic sheaths of the vessels may be filled with white blood corpuscles.

Occasionally a diffuse opacity of the retina is met with, said by Roth to be due to a thickening of the vertical fibres of the retina, but probably sometimes due to œdema—of the ganglion-cell layer in the case described by Deutschmann,[1] of both ganglion-cell and nerve-fibre layers in a case examined by Oeller,[3] in which both these layers were twice the normal thickness. When considerable it is in part due to a diffuse infiltration of leucocytes, as described by Poncet and Oeller. This opacity, with some swelling and great tortuosity of the veins, was the chief appearance in one case under my own observation. The patient was a woman, aged thirty-six, in University College Hospital, under the care of Dr. Wilson Fox. Both eyes were affected in a similar manner.

[1] "Kl. Monatsbl. für Augenheilk.," 1887, p. 231.
[2] Such capillaries are figured by Poncet, "Atlas," pl. 66.
[3] "Arch. f. Ophth.," xxiv. 1878, pt. iii. 241.

The discs were clear, the sclerotic ring distinct, and the physiological cup quite normal. The tint of the periphery of the disc was rather deep, but there was no punctiform redness. There was a diffuse, slight opacity of the retina, chiefly marked near the disc and somewhat striated on direct examination. Towards the periphery of the retina, a few small white spots were seen, and one small hæmorrhage. The arteries were nearly normal in size, but unduly tortuous. The veins were greatly increased in diameter, and much paler than natural. Their central reflection was everywhere distinct and broad. The smaller veins were conspicuous and could be followed for a longer distance than normal. The larger veins were very tortuous, the curves being chiefly in the plane of the retina, but some antero-posterior. One or two small veins on leaving the disc were lost in the opacity of the retina, but the larger veins were not concealed.

A remarkable change was observed by Heinzel,[1] in a case of lymphatico-splenic leucocythæmia in a child, 4½ years of age. There was at first an enormous swelling of both papillæ, which were occupied by a striated opacity, completely concealing the disc, without redness, ceasing two discs' breadth from the edge. The retina presented here and there a little opacity. There was moderate tortuosity of the veins, and the vessels were bordered by pale lines of variable width. Numerous hæmorrhages appeared and disappeared in each retina. All the pathological appearances passed away in four weeks, the fundus appearing normal but pale, and it was also found normal after death. Heinzel remarks that the appearance had not the aspect of an inflammatory process, but rather that of mechanical congestion with consequent œdema and ecchymoses. Such a condition was presented in the same case by the conjunctiva, and several times by the skin, and was ascribed to the constitutional state.[2]

In cases of general thickening of the retina, the optic

[1] "Jahrbuch für Kinderheilk.," 1875, p. 346.

[2] The ophthalmoscopic appearances may have been due to thrombosis in the orbital vein, the anastomoses with the facial ultimately sufficing to restore the normal circulation. Venous thrombosis is common in this disease.

papilla has been found swollen (to ·3 mm.—Oeller) in consequence of leucocytal infiltration and œdema. The leucocytes were densely massed in front of the lamina cribrosa. The disc is not usually changed, however, except when the parenchymatous retinal changes are considerable.

The hæmorrhages may be, in some cases of leucocythæmia, so numerous as to give to the changes the aspect of a hæmorrhagic retinitis, such as is met with occasionally in other conditions. A large extravasation may burst through into the vitreous, and cause secondary glaucoma. The vessels are sometimes accompanied with conspicuous white lines.

Besides the changes which appear related to the blood-state, the complication of kidney disease may lead to retinal changes, identical in appearance and structure with those met with in cases of primary renal disease, as in a case figured by Poncet.[1]

The degree to which the changes interfere with sight depends on their extent and position. If abundant they cause considerable amblyopia; if slight the vision may be unimpaired, and the retinal changes may easily be overlooked, unless systematic examination is made with the ophthalmoscope. In a case recorded by Hirschberg[2] the patient sought advice in consequence of seeing a red balloon constantly before his right eye, and the ophthalmoscope showed a large hæmorrhage of corresponding shape in the macular region, with numerous small ones scattered over the fundus. His blood and spleen were examined, and the diagnosis of leucocythæmia confirmed. A month later, an exactly similar hæmorrhage occurred in the left eye, giving rise to a spectral red balloon in that eye also. When situated near the macula, central vision is much impaired; in the case figured, for instance (Pl. XII. 2), it was very dim, but not lost. Occasionally the disturbance of the retinal elements leads to a curious change in vision, as in one case in which parallel lines appeared to come near together, and again to

[1] Perrin and Poncet: "Atlas," pl. 66.

[2] "Centr. f. prakt. Augenh.," 1887, p. 97; "Ophth. Rev.," 1888, p. 12.

diverge. Double exophthalmos, from a lymphoid growth in both orbits, was present in a remarkable case of leucocythæmia described by Leber.[1] Retinal hæmorrhages were also present. A case of exophthalmos in this disease, described by Chauvel,[2] was probably of the same nature.

Sometimes the choroid is found infiltrated with leucocyte-like cells, and its vessels may be, at the same time, greatly dilated. From these two changes, in Oeller's case, the choroid, near the outer side of the disc, was swollen to eight times the normal thickness. It was difficult to say whether the leucocytes were free or were contained in the enormously dilated vessels. Poncet has figured an infiltration of the iris with leucocytes, supposed to indicate a leucocythæmic iritis.

Purpura.

The tendency to rupture of vessels in purpura leads to retinal as well as to subcutaneous extravasation. How frequently retinal hæmorrhages occur cannot be ascertained until the ophthalmoscope is more generally used by physicians, but they are certainly very common, perhaps invariable in the severer forms of the affection. Cases have been recorded by Ruc,[3] Stephen Mackenzie,[4] and others. In each of two fatal cases recently under the care of Dr. Hunt, late of Wolverhampton, retinal hæmorrhages were present and numerous.[5] The extravasations are for the most part striated, and adjacent to vessels, and most abundant in the neighbourhood of the optic disc. In a case recorded by Goodhart,[6] a large subretinal extravasation was present, and was thought to be in the choroid. It had a white edge, and white spots have been seen in connection with the retinal hæmorrhages in this as in other affections. The occurrence

[1] "Arch. f. Ophth.," vol. xxiv. 1878, p. 295.
[2] "Gaz. Hebd.," 1877, No. 23.
[3] "L'Union Méd.," 1870.
[4] "Med. Times and Gaz.," 1877 292.
[5] Oral communication.
[6] "Lancet," 1878, i. p. 123.

of extravasations into the retina indicates a severe, but not necessarily fatal, degree of the disease. The hæmorrhages may disappear, and be replaced by others, and the patient may ultimately recover. Hæmorrhage into the choroid was also found post-mortem by Ruc, in a case in which a large number of retinal extravasations were observed during life. The latter may cause considerable amblyopia if numerous, and, as in other cases, if near the macula lutea, may damage central vision.

Scurvy.

Retinal hæmorrhages have been found in scurvy, but less commonly than in purpura; perhaps because they have not been looked for. They are, as in purpura, commonly in the neighbourhood of the optic nerve. In one case recorded by Wegscheider,[1] numerous small extravasations into the brain co-existed.

DISEASES OF THE LUNGS.

Pulmonary affections rarely cause ocular troubles. Emphysema of the lungs may lead to mechanical congestion of the venous system generally, which may be conspicuous in the eye. The same influence has been ascribed, but on very doubtful grounds, to phthisis, in which amblyopia occasionally occurs. Schmall mentions that he has often seen a more or less lively injection of the fundus in this disease, and that he found visible arterial pulsation in five cases.[2] Tubercles in the choroid may be met with in cases of acute tuberculosis, but never when the tubercular affection is confined to the lungs. Acute pneumonia is said, in one case, to have been associated with neuro-retinitis. A febrile intense bronchial catarrh in a young woman, with much cyanosis, was observed by Litten[3] to be accompanied with

1 "Deutsche Med. Wochenschr.," Nos. 17 and 18, 1877.
2 "Graefe's Archiv.," xxxiv. i. p. 37; "Ophth. Rev.," 1888, p. 268.
3 "Charité Annalen" for 1876. Berlin, 1878.

neuro-retinitis, in and around the papilla, of gradual development, and with numerous extravasations, some with white centres, near the equator of the eye. Many of the extravasations were regularly arranged, and situated upon small veins; and he suggests that the changes were probably set up by the great distension of the veins. The retinal affection subsided with the bronchitis.

DISEASES OF THE DIGESTIVE ORGANS.

The occasional effect of hæmorrhage from the stomach and intestine has been already described. Galezowski [1] associates atrophy of the optic nerve, in some cases, with chronic gastric troubles. He has described several remarkable cases in which great improvement occurred in amblyopia, previously obstinate, on correcting gastric or intestinal troubles. In some cases there was also tenderness of the lower cervical spine. Chronic diarrhœa may also, in the opinion of the same author, lead to a "perivascular retinitis," in which an infiltration of the retina, causing opacity, may extend around the vessels. The association of these conditions has not, however, been generally recognized.

Constipation is regarded by Eales[2] as having been influential in causing retinal hæmorrhage in a series of cases observed by him. All were young men, with slow pulse and high arterial tension, and two had a slight trace of albumen in the urine. The extravasations were chiefly in the left retina, roundish in form as if in the deeper layers. He speculates that the constipation may have been due to or accompanied by vaso-motor spasm in the abdominal vessels, sufficient to cause a general increase of arterial tension.

Jaundice.—The changes in the blood from jaundice, from any cause, may occasion retinal hæmorrhage.[3] Jäger says

[1] "Journ. d'Ophthalmologie," March, 1872. "L'Union Méd.," 1876, i. p. 368.

[2] "Birmingham Medical Review," July, 1880, p. 262.

[3] Litten: "Zeitsch. f. Klin. Med.," v. i. p. 319.

that the blood in the vessels may have a yellowish tint, but the appearance is probably due to a tint in the media, such as in rare cases causes yellow vision.

DISEASES OF THE SEXUAL ORGANS.

Sexual excess in men has been said to cause atrophy of the optic nerve, but the evidence in support of the relation of the two is not strong.

Sudden suppression of the menses has been observed to be followed by acute optic neuritis, such as accompanies meningitis, and often attended with unpleasant sensations in the head. The occurrence of the neuritis is probably analogous to the occasional production of other acute changes in the nervous system, such as acute myelitis, from the same cause.

In chronic menstrual irregularities, optic neuritis, of chronic course, has been found, and occasionally other disturbances, such as retinal hæmorrhages. It is probable that in most cases of this character the two conditions—the ocular and menstrual disturbance—are related to a common cause. In a case recorded by Spencer Watson retinal hæmorrhages with high arterial tension occurred at the climacteric period (see p. 28).

The occurrence of loss of sight, sometimes with neuritis, after uterine hæmorrhage has been already mentioned (p. 237).

In pregnancy, albuminuric retinitis is occasionally developed. Under the title "amaurosis by reflex irritation," Landesberg[1] has related two remarkable cases of amblyopia, with limitation of field, coming on in pregnancy. In one case the affection of sight quickly passed away; in the other it was accompanied by hemianæsthesia, and cyclitis developed, which necessitated enucleation. A similar condition has been observed, apparently due to menorrhagia. The nature of these cases is obscure. Although most cases of affection of sight from pregnancy are produced through the agency of albuminuric retinitis, it would seem that

[1] "Arch. f. Ophth.," xxiv. pt. 1, p. 161.

more direct influence is sometimes exerted. Loring[1] has described a case in which each of three pregnancies was accompanied with failure of sight, the first two in the outer half of one field, the last in the outer half of each, with general impairment of vision.

DISEASES OF THE SKIN.

It has been said by some, especially by Mooren, that general skin diseases may be accompanied by inflammation of the retina and papilla; that eczema of the head may be accompanied by optic neuritis, ending in atrophy. The statement has, however, received no confirmation, and the relation of the two conditions must be considered as exceedingly doubtful. If such a sequence occurs, it is possibly by the production of a local orbital cellulitis. The suppression of a customary cutaneous discharge, such as that of eczema, has also been said to cause neuritis, but the statement needs corroboration.

A case of slow atrophy of both optic nerves, associated with a general herpetic eruption on the skin, diagnosed by Hebra as "chronic herpes zoster," has been recorded by Hubsch.[2] But the patient passed through a condition of delirium to one of imbecility, and the atrophy was probably part of a widely spread degeneration of the nervous system, of which the skin eruption may have been an effect.

CHRONIC GENERAL DISEASES.

Tuberculosis.

The grey granulations which constitute the anatomical lesion in tuberculosis may form in the vascular structures of the eye, chiefly in the choroid, rarely in the iris and retina.

[1] "New York Med. Jour.," 1883, p. 59.
[2] "Ann. d'Oculist.," 1872, p. 239.

When present in the fundus, they may readily be seen with the ophthalmoscope.[1]

Tubercles in the choroid (Fig. 77) appear to the ophthalmoscope as white, yellowish-white, or reddish-yellow spots, usually isolated, and more or less rounded in form. They are palest in the centre, and commonly redder on their outer portions, and the peripheral redness passes gradually into that of the adjacent choroid. They commonly develope in the substance of the choroid, and the pigment and vessels atrophy before the growing nodule, first, and more completely, at the centre, so that the diameter of the tubercle on section may be found to be twice or three times as great as that of its exposed portion (Fig. 78). In size they vary from one-third of a millimetre, to two, or two and a half millimetres, *i.e.*, from about one-fourth to half or three-quarters the diameter of the optic disc. The larger sizes are rare. Occasionally several are aggregated together to form a mass which may be the size of the disc or even larger—seven or eight millimetres in

FIG. 77.

FIGS. 77—79.—TUBERCLES OF THE CHOROID FROM A CASE OF ACUTE MILIARY TUBERCULOSIS IN A CHILD.

FIG. 77.—The front of the eye has been removed, and the retina is drawn over to the left. Six tubercles are seen, varying in diameter from 1 to 4 mm. (× 2). They are prominent in the darker periphery, the pigment-epithelium over the choroid is intact, and where it has disappeared the tubercle appears within.

[1] That tubercles occurred in the choroid as a post-mortem observation has long been known. They were described by Autenrieth in 1808. They were first observed with the ophthalmoscope by Ed. Jäger in 1855.

diameter. These larger masses project considerably into the eye. Slight prominence may commonly be recognized in all the larger tubercles, and assists the diagnosis. The smaller ones may resemble spots of choroidal exudation or atrophy. From the former, their rounded shape and yellowish tint distinguish them. From atrophy, the tint, regular form, concealment of the choroidal vessels, and the (common) absence of any adjacent pigmentary disturbance are sufficient distinctions. They are plainly behind the retinal vessels. In structure they consist of the same lymphoid cells as constitute the granulations elsewhere. The cells are distinct in the periphery, degenerated in the centre (Fig. 79). Extravasations of blood existed in the substance of a granulation in one case examined by myself. The tubercles are situated chiefly at the middle of the fundus,

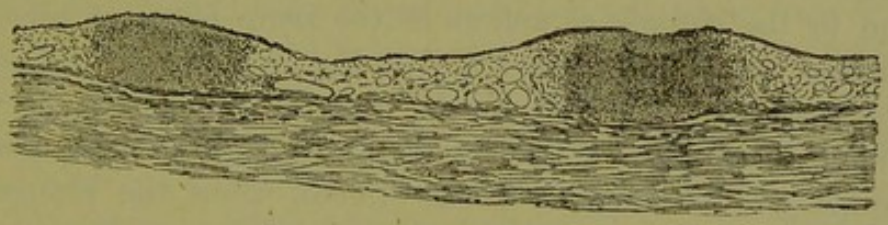


FIG. 78.

FIG. 78.—Section of two of the smaller of these tubercles. They occupy the whole thickness of the vascular layer, pushing forward the pigment-epithelium, and, in the case of the right hand one, breaking through it. (× 30.)

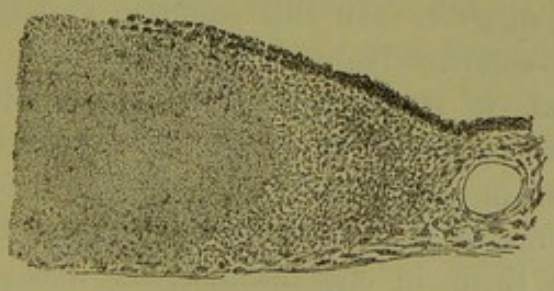


FIG. 79.

FIG. 79.—One half of a tubercle, which has caseated. Above it, in the centre, where it has a granular aspect, the pigment-epithelium has disappeared towards the centre, and below are the deeper pigment cells of the choroid. (× 100.)

not far from the optic nerve entrance. Usually only three or four are present; sometimes, however, as many as twelve or twenty or even fifty (Cohnheim). They may form rapidly, and, according to Stricker, may become recognizable in from twelve to twenty-four hours. But it must be remembered that they attain a considerable size, without disturbance of the epithelium, and the partial removal of this may rapidly increase their distinctness.

The tubercles begin as minute points, "masses of lymphoid cells," and develope in the structure of the choroid, advancing towards the retina until they cause atrophy of the pigmentary layer over the choroid, and become ophthalmoscopically visible. When the tubercles are large and grow rapidly, the pigment frequently remains on their surface in the form of spots.

Choroidal tubercles occur in both children and adults, and in the chronic as well as in the acute forms of tuberculosis, but are most frequent in the acute forms. They are practically confined to the cases in which tubercle is widely distributed. Their actual frequency in these cases cannot yet be stated. It is evident, from the rapidity of their appearance, that repeated ophthalmoscopic examination is necessary to exclude their occurrence. Cohnheim described them as very commonly to be found after death, and Litten found them (post-mortem) in thirty-nine out of fifty-two cases. According to most observers they are much less frequently to be seen during life than these figures would suggest. In this country, at any rate, they appear to be comparatively rare. As a rule, when they are discovered, it is not until the disease has become advanced. Exceptions to this have, however, been recorded, as in one case in which they were present before fever or other symptoms of the disease were developed.[1] Steffen,[2] again, found them, in one instance, six weeks before the commencement of tubercular meningitis. In such a case

[1] Fraenkel: "Berl. Kl. Wochenschr.," 1872; "Jahrbuch für Kinderheilk.," Bd. ii.

[2] "Jahrbuch für Kinderheilk.," 1870.

they may afford great assistance to diagnosis. Their presence, then, is of value as evidence of general tuberculosis, especially in cases in which the diagnosis of acute tuberculosis from other acute febrile conditions is difficult; their absence is of no significance. Tubercles of the choroid often coincide with tubercular meningitis, but in a few cases they have been met with when the membranes were free from tubercle. It is remarkable that the characteristic bacilli cannot be found in many cases, although they have been readily detected in the tubercles of the membranes in the same subjects. Thus out of six cases examined by Lawford, they could not be detected in four, after the most thorough investigation in various ways, while in the other two he found them with ease.[1] Haab, however, found them almost invariably.[2] In a case of choroidal tubercle, where bacilli could not be demonstrated, the inoculation of a guinea-pig with the crushed tubercle produced general tuberculosis.[3]

As a rule choroidal tubercles cause no symptoms. Transient disturbances in sight have, however, been described. In a case related by Manz[4] tubercular growths perforated the sclerotic and appeared on the exterior of the eye.

Occasionally, although rarely, a tubercular mass developes in the deeper structures of the eye, quite similar to the masses of the same nature which are found in the brain. One case, in which such growths were associated in these two situations, has come under my observation; optic neuritis was also present. A tubercular mass, with granulations in the neighbourhood, infiltrated the greater part of one optic nerve and invaded the eye, in a case described by Chiari,[5] appearing as a white prominence in the position of the papilla, five disc-diameters in width.

[1] "Trans. Ophth. Soc.," vi. p. 348, where a summary of the results obtained by various observers will be found.

[2] "Klin. Monatsbl. für Augen.," 1884, p. 391.

[3] Alexander: "Centralb. f. Augenh.," 1884, p. 161.

[4] "Klin. Monatsbl.," Jan. 1881, p. 26.

[5] "Wien. Med. Jahrbuch," 1877, p. 559. Sattler: "Arch. f. Ophth.,' Bd. xxiv. pt. iii. p. 127.

Choroidal tubercles were found by Cohnheim in a guinea-pig rendered tubercular by inoculation.

Retina.—The occurrence of tubercles of the retina has been recorded in very rare instances. The aggregations of lymphoid cells which may occur in the nuclear and molecular layers, adjacent to an inflamed disc in tubercular meningitis, have been regarded as such, but their tubercular nature is uncertain. Unequivocal tubercles in the retina (often containing giant cells) have usually been associated with tubercular growths in almost all the structures of the eye (Perls, Manfredi), in rare cases with a tubercular papillitis only (Weiss, Sattler). In the case of tubercle of the optic nerve referred to above, the optic papilla was the seat of a large mass of caseating tubercle, and miliary tubercles were scattered through all the layers of the adjacent retina.[1]

Tubercles in the eye are, as already stated, almost invariably part of general tuberculosis. In one case, however, they were found in all parts of the eye, although absent elsewhere.[2]

Local deposits of tubercle in the encephalon may, as already described, give rise to ophthalmoscopic changes, producing optic neuritis, as do other cerebral tumours. In rare instances tubercular masses are situated in the intracranial portion of the optic nerves[3] or in the chiasma,[4] and may cause a corresponding affection of sight (probably with or without evidence of descending neuritis). The inflammation which accompanies the formation of tubercle in the meninges may also be accompanied by neuritic changes in the eye (see p. 173).

In tuberculosis of the brain of guinea-pigs, artificially produced, Deutschmann[5] has found a development of tubercles in the sheaths of the optic nerves close to the eye, accompanied, during life, by slight papillitis. The disease

[1] "Arch. f. Ophth.," xxiv. pt. iii. p. 150.
[2] Weiss: "Arch. f. Ophth.," xxiii. pt. iv. p. 57.
[3] Cruveilhier: "Anat. Path. Gén.," 1862, Bd. iv.
[4] Hjort: "Kl. Monatsbl.," 1867, p. 166.
[5] "Arch. f. Ophth.," xxvii. pt. i. p. 251.

appeared to have resulted, not by direct continuity with that in the brain, but by the passage of a *materies morbi* into the sheath, and its arrest at the anterior extremity of the vaginal space. Changes at the same spot, apparently tubercular, and accompanied by a perineuritis and some interstitial neuritis, were found in a child who had died of tubercular meningitis.[1] The changes ceased a centimetre from the eye. There were no ophthalmoscopic changes.

Tubercular disease of the lungs has been described by some observers as accompanied by mechanical congestion of the retinal veins and by disturbed vision, but it is very doubtful whether these are in any cases related to the pulmonary affection.

Morbid Growths.

Morbid growths other than tubercular are very seldom present at the same time in the eye and brain. Cancer of both choroids has, however, been observed by Puts, occurring secondarily to a primary epithelioma of the lung.

Syphilis.

Acquired Syphilis.—The syphilitic diseases of the eye, during their active stage, commonly come under the care of the ophthalmic surgeon. Their consequences in the fundus oculi are, however, among the appearances which the physician encounters most frequently in his own work, and which often furnish him with very useful information. A knowledge of these changes is, therefore, of great importance.

Iris.—Although not strictly an ophthalmoscopic sign, the evidence of a past attack of iritis is often first discovered by the ophthalmoscope revealing the presence of uveal pigment on the anterior surface of the lens. In the majority of cases iritis is due to, and its traces are signs of, constitutional

[1] "Virch. Arch.," lvi. p. 497.

syphilis; and the importance of the evidence thus afforded, from its frequency and easy recognition, can hardly be over-rated. Over and over again it must have occurred to all physicians who use the ophthalmoscope, when looking for changes in the fundus oculi, to encounter these signs of past iritis, in cases in which syphilis had not been suspected, and often in which no other indication of it, in history or symptoms, was to be obtained.

Choroid.—Next to the iris, the choroid is affected by syphilitic disease more frequently than any other part of the eye. In spite of the opinions which have been expressed by some authorities, there is strong reason to believe that disseminated choroiditis is, in the great majority of cases, syphilitic. In its active stage, in which extensive areas of white exudation, comparable, in Hutchinson's opinion, to gummata, are the conspicuous features, it rarely comes under the notice of the physician. In its later stage, in which extensive regions of atrophy alternate with scattered stellate and crater-like pigmentary deposits, and sometimes with hæmorrhages, it is often met with. It is to be remembered that the pigment is deposited in the retina as well, and care must be taken to avoid confounding the change with primary retinitis pigmentosa. The choroiditis of acquired syphilis may be either unilateral or bilateral. When slight, the traces of it may be detected only in the peripheral part of the retina towards the ora serrata.

Retina.—Isolated syphilitic retinitis is less common than syphilitic choroiditis. It is characterized by areas of diffuse opacity, parenchymatous swelling, tortuous vessels, and a blurred disc. The vitreous frequently shows fine dust-like opacities. Sight is considerably impaired. This form also comes chiefly under the ophthalmic surgeon's notice.

Optic Nerve.—Neuritis, limited to the papilla, is common in syphilis, as secondary to brain disease, but is very rare as a primary syphilitic affection, if it ever occurs. (See pp. 151, 152.) Neuritis has, however, been met with secondary to a syphiloma of the trunk of the optic nerve.

Simple atrophy of the nerve is said occasionally to occur,

as the consequence of syphilis, double, unaccompanied by spinal symptoms (Galezowski).[1] The occurrence of atrophy from this cause is not surprising, since there is reason to believe that constitutional syphilis is a powerful predisponent to locomotor ataxy, in its purely degenerative form. It is probable that one-half the patients with ataxy would not suffer from the disease had they not at some previous time suffered from syphilis.[2] This is true of cases with, as well as of those without, optic nerve atrophy. Moreover, the loss of reflex action of the iris, which so commonly accompanies ataxy, may occur without spinal symptoms in cases of constitutional syphilis, as in several cases which I have seen. One of them presented also optic nerve atrophy, similar to that which accompanies spinal disease; but of such disease there were no symptoms; even the knee-jerk was normal.

Inherited Syphilis.—The characteristic indication of inherited syphilis which is afforded by interstitial inflammation of the cornea, and a knowledge of which we owe to Mr. Hutchinson, is well known, and does not come within the scope of the present work.

Of the deeper structures of the eye, the one most liable to be affected is the choroid, which is often the seat of disseminated inflammation, in infancy or later. Scattered areas of atrophy may be left, associated with accumulations of pigment, just as in the form which results from the acquired disease. When slight, small round spots of atrophy may be seen, surrounded with pigment. This form is very characteristic, although rare, and, when the pigment is slight, is sometimes, as I have seen, mistaken for tubercles of the choroid. The course of the choroiditis is well illustrated by two cases described by T. Barlow,[3] in one of which the autopsy showed also chronic syphilitic disease of the cerebral

[1] "Journ. d'Ophthalmologie," March, 1872, p. 139.

[2] For the evidence on which this statement is made, see "Syphilis and Locomotor Ataxy," "Lancet," Jan. 1881, p. 94, and the statistics of Erb there referred to, and also those brought by him before the International Medical Congress, 1881.

[3] "Trans. Path. Soc.," 1877, p. 287.

membranes. The choroid presented, in each case, brownish flecks of exudation without disturbance of pigment or atrophy. The microscopical examination (by Nettleship) showed the chorio-capillaris beneath these flecks to be infiltrated with pus-like cells, and in several instances there was a layer of flattened cells on the surface next to the retina. In the other case Barlow traced the progress of similar flecks to a stage of atrophy, such as is seen in disseminated choroiditis—the condition occasionally met with later on in life. Several cases of this character have been recorded by Hutchinson.[1]

A peculiar form of atrophy of the disc has several times come under my notice in children the subjects of this disease. The disc has a uniform reddish tint, the edges are not well defined, and the vessels are small. There has not commonly been any atrophy of the choroid or pigmentary accumulation. It is probably secondary to retinitis or widespread capillary choroiditis. Several times since first observing the connection between the two conditions, this form of atrophy has drawn my attention to the existence of inherited syphilis, which had otherwise escaped notice. Sight is usually impaired, sometimes considerably.

Retinitis sometimes occurs in the inherited just as in the acquired disease.

Retinitis pigmentosa is, by some, believed to be connected with inherited syphilis, and an instance of the association of the two diseases has been described by Swanzy. Deposits of pigment in the retina occasionally accompany the atrophic changes in disseminated choroiditis, but the connection of true retinitis pigmentosa with syphilis is generally considered to be very doubtful.

[1] One is figured in the "Ophth. Hosp. Rep.," vii. Pl. 4, Fig. 3. Mr. Hutchinson has suggested that the condition of choroidal atrophy and choroidal and retinal pigmentation may occur without an exudative stage.

Chronic Rheumatism.

Chronic rheumatism has only accidental associations with changes in the fundus oculi. Neuro-retinitis has been ascribed to "rheumatism." Probably some of the cases were instances of gout, with albuminuric retinitis due to granular kidneys. It must be remembered also that the optic nerve, like the other orbital nerves, may be damaged by rheumatic inflammation at the back of the orbit (see p. 182).

Gout.

The influence of gout in producing kidney disease renders it a powerful indirect cause of the retinal affection which accompanies albuminuria. There are, however, other ophthalmoscopic changes which are to be ascribed with more or less probability to the gouty state of the blood. They are seldom seen in corresponding general conditions, except in persons who are the subjects of gout, so that they become important as evidence of a constitutional state which may have been previously obscure.

(1.) *Hæmorrhagic Retinitis.*—The frequency with which the subjects of this affection present a history of gout was first pointed out by Hutchinson,[1] and the influence seems well established, although its extent is possibly exaggerated. The extravasations may be small, flame-shaped, and scattered over the whole fundus. They are usually present in one eye only, often the left. Hæmorrhages may recur for a long period. Hutchinson believes that they may occur in young persons from inherited gout. He suggested that their cause might be thrombosis in the central retinal vein, from which Michel has shown that extensive hæmorrhages may result (see p. 31). The obstruction in the vein is probably only a very partial one, however, in many of the cases, since there is no such intense nervous engorgement as has been found

[1] "Trans. Clin. Soc.," vol. xi. p. 132. See also "Trans. Ophth. Soc.," vol. i. 1881, p. 26.

when the thrombosis was nearly or quite complete. Again, the well-known tendency to recurrence of the extravasations is in favour of the cause being a partial obstruction, such as would result from the gradual formation of a parietal thrombus at one or more points in the vein, in connection with changes in or around its walls.

Galezowski has also described hæmorrhages leaving white patches of "sclerosis" as occurring in gouty persons.

(2.) *Retro-bulbar Neuritis.*—We have learned that spontaneous inflammation of nerve trunks and plexuses, on one side only, and occurring after the age of thirty or forty, is seldom due to any other cause than gout. Such neuritis means always primary perineuritis. This would lead us to expect that such an influence would be exerted frequently on a nerve so prone to suffer from inflammation as the optic, but inflammation of its sheath behind the eye is less common in ordinary gout than might be anticipated. Acute or subacute inflammation is, however, met with in young adults in whom no other cause can be traced, and in whom this cause is never absent,—facts that confirm the relation to inherited gout long maintained by Mr. Hutchinson. The characteristics of these cases are, of course, the greater degree of affection of sight than corresponds to the visible changes in the optic disc; the tendency to irregular defects in the field of vision; and the strong tendency to the affection of the second nerve, not by extension through the commissure, but by an independent symmetrical morbid process. This always proves a general cause. Occasionally the chiasma is invaded. The part of the nerve diseased differs, and with it the affection of sight varies. Probably, in some cases, the affection commences at the chiasma, and simulates compression by a tumour. When the process is far back, no signs of inflammation may be seen within the eye, or only such as are slight and equivocal, but atrophy slowly supervenes.

There is often much pain—for the subjects are commonly prone to neuralgia. As a rule other orbital nerves escape—a contrast to the rheumatic cellulitis and perineuritis mentioned above (p. 182).

Lead Poisoning.

The eye is occasionally affected in lead poisoning, apart from the effects of induced kidney disease. It may suffer in three ways. There may be (1) amblyopia, usually transient, without ophthalmoscopic changes; (2) atrophy of the optic nerve; (3) optic neuritis.

The occurrence of blindness in lead poisoning has long been known. Some well-marked cases were published by Duplay in 1834.[1] In what v. Graefe called "the pre-ophthalmoscopic period," the transient amblyopia attracted, however, more attention than the graver forms of affection, so that Tanquerel des Planches spoke of the amblyopia as almost invariably passing away. Optic nerve atrophy in lead poisoning was first described by Hirschler in 1866,[2] and optic neuritis by Meyer in 1868.[3] Attention was, however, especially called to the affection by the publication of an important series of cases by Hutchinson in 1871.[4]

(1.) The transient amblyopia, without ophthalmoscopic changes, is usually sudden in onset, and may be complete. It has been observed in some cases of acute saturnism after but short exposure to the exciting cause. It commonly soon passes away, and is probably due to a direct effect of the lead on the nerve centres, analogous to the temporary amaurosis of uræmia and diabetes. In one case recorded by Fano there was, for some months, a periodical transient failure of sight at the same hour each day.

Hemianæsthesia has been observed (chiefly on the Continent) in consequence of lead poisoning. It is apparently of functional origin, and may be due to the same mechanism as the transient amblyopia. The two coincided in a remarkable case which has been recorded by Landolt,[5] although in

[1] "Arch. Gén. de Méd.," 1834.

[2] "Wien. Med. Wochenschr.," 1866, Nos. 6 and 7. It is not easy to say, from the account of the case, whether neuritis was present or not. The disc is spoken of as being grey and having lost its transparency.

[3] "L'Union Méd.," No. 78.

[4] "Ophth. Hosp. Rep.," vol. vii. p. 6.

[5] "Ann. d'Oculistique," vol. lxxxiii. March, 1880, p. 165.

this case it is very doubtful whether the symptoms were due entirely to functional disturbance. Hemiplegic weakness, hemianæsthesia, and amblyopia came on together. The loss of power lessened, but the loss of sensibility and affection of sight persisted. Six months later, the field of vision of the eye on the affected side presented slight peripheral limitation and a central scotoma with complete loss of colour-vision. The other field presented two concentric annular scotomata with partial loss of colour-vision, blue, violet, and yellow being lost, red and green not lost. Acuity in each was reduced to counting fingers. The discs were merely greyish red in tint, the veins large. The application of an electro-magnet is said to have restored sensibility to the side, to have restored colour-vision, and to have improved acuity, but the scotomata remained till the patient resumed his work and was lost sight of.

Stood[1] has published several cases where there was progressive concentric contraction of both fields, both for white light and for colours. Sometimes there was slight neuritis. Central scotoma was present in a few cases only.

(2.) In amblyopia of long duration it is common to find the signs of atrophy of the optic nerves. The discs are sharp-edged, pale, and often greyish, the arteries small. It is said that the atrophy may be from the first unattended by vascular changes (Horner). In a considerable number of cases, however, which have been seen in an early stage, a condition of simple congestion of the discs has been found. The tint is uniformly red, with softened edges, with little or no swelling. Gradually the redness fades, and a reddish-grey atrophy results, often with distinct white lines along the narrowed vessels. Sight has been much affected in all the recorded cases, the acuity of vision impaired, and the field presenting a central or peripheral defect. The loss commonly progresses until even quantitative perception of light may be lost. This congestive atrophy is usually double, but, as a case recorded by Hutchinson shows, the affection of one eye may precede that of the other.

[1] "Arch. f. Ophth.," 1884, iii. p. 215.

(3.) Occasionally cases of lead poisoning present much more pronounced inflammatory changes—considerable papillitis with swelling, obscuration of the edge of the disc, concealment of vessels, and hæmorrhages, especially at the margin of the swelling. The arteries beyond the swelling are commonly narrow; the veins may be distended or of normal size. An instance of this form of neuritis is shown at Pl. VII. 6. The affection is almost always double, and usually entails considerable amblyopia. There is reason to believe, however, that slight degrees of neuritis are not uncommon in lead poisoning without any affection of sight. I have seen one such case, and the general use of the ophthalmoscope in medical practice will probably show it to be not an infrequent occurrence. Pronounced neuritis may subside into atrophy having the characters of "consecutive atrophy," leaving a dull-white, full-looking disc, with narrow arteries. Or the neuritis may clear and sight be recovered. In a case recorded by Schrœder, there was well-marked papillitis with hæmorrhages, without any pronounced cerebral symptoms, although there was some palsy of both sixth nerves. There was no central scotoma. Rapid improvement occurred under treatment. It is important to remember that the affection of sight in these cases may be in part due to the direct effect of the lead on the nervous system, which has been already mentioned. This is the more likely when, as in many recorded cases, loss of sight comes on suddenly. The transient amblyopia, mentioned above, has been noted in association with neuritis[1] as well as with normal ophthalmoscopic appearances.

These ocular changes commonly occur in chronic cases of lead poisoning, which have presented toxic symptoms for some time, often for years, previously, and they may coincide with an increase of the other symptoms. It must be remembered, however, that the manifestations of lead are very irregular, and any one may be absent or may alone be present. In cases of very recent intense lead poisoning,

[1] A case reported by Stricker from Traube's Clinique, and quoted by Abadie ("Mal. des Yeux").

toxic amblyopia and neuritis are apparently more common than atrophy. A case in which neuritis, going on to atrophy, occurred in a girl of seventeen, after four years' work in a type foundry, has been recorded by Hirschberg.[1] She suffered also from various nerve-troubles.

In many cases of lead poisoning the occurrence of neuritis coincides with symptoms of cerebral disturbance, headache, convulsions, delirium,[2] &c. The case figured in Pl. VII. 6 presented, at the same time as the neuritis, much mental disturbance, and both symptoms passed away together. In a fatal case of this description, recorded by R. Atkinson,[3] there were no naked-eye changes in the meninges or brain, but lead was found in it in quantity equivalent to five grains in the whole brain. This association of cerebral disturbance with optic neuritis in these cases is probably more than a coincidence, and the analogous fact as regards albuminuric neuritis (p. 222) may be borne in mind.

The diagnosis of saturnine atrophy and neuritis rests especially on the recognition of the signs of lead poisoning, the line on the gums, the occurrence of gout, of colic, of wrist-drop, and the presence of anæmia. It is only by these symptoms that the neuritis can be distinguished from that of primary encephalic affections. The possibility of a renal neuritis in cases of lead poisoning must be borne in mind. It is highly probable that in at least one published case the retinal change was due to the albuminuria rather than to the lead. Albuminuric retinitis is not uncommon in cases of chronic lead poisoning of long duration. Again, an intracranial syphilitic growth may coexist with lead poisoning, and give rise to ocular symptoms, and as the ordinary treatment for lead poisoning may cause the disappearance of the syphilitic lesion, the error of attributing the ocular conditions to the toxic influence of the lead may very easily be made.

[1] "Arch. f. Augenkrankheiten," 1879, p. 9.

[2] This is an old observation. Tanquerel des Planches described saturnine amaurosis as the accompaniment of encephalopathia and colica saturnina, and stated that in cases of this kind no material change is to be found in the brain ("Traité des Mal. de Plomb.," 1839, tom. ii. pp. 211 and 235).

[3] "Lancet," 1878, i. p. 784.

The prognosis in all forms of change in the optic disc must be cautious. It is least grave in the case of toxic amblyopia, next in cases of pronounced neuritis, especially of acute course, less so in cases of chronic congestive change, and in pronounced atrophy it is very unfavourable. Of fourteen cases of various forms, collected by Lespille-Moutard,[1] nine progressed to blindness.

The treatment is essentially that for the general state, but local applications, leeching and counter-irritation, have appeared useful in some cases.

Chronic Alcoholism.

Atrophy of the optic discs, sometimes of one only, sometimes of both, is occasionally met with in the subjects of chronic alcoholism, to which it is apparently due. It is said to occur especially in sedentary drinkers, to be more common on the Continent than in this country, and to result from spirit-drinking rather than from wine. Hence, according to Rominée,[2] it is much more common in the north of France, where much cheap brandy is consumed, than in the wine-producing districts of the south. Amblyopia may precede any ophthalmoscopic change, and is characterized by a central dimness or defect (Förster), very similar to that met with from tobacco,[3] but said to be more exactly central (Hirschberg). Before there is recognizable defect for white light, a defect for red may be discovered, extending from the fixing point to the blind spot and a little beyond each. To detect it in slight cases the ordinary colour tests do not suffice, since the coloured object should not be more than five millimetres in diameter. Extensive loss of vision for certain colours, as green and violet, has also been described by

[1] "Thèse de Paris," 1878. [2] "Recueil d'Ophth., 1881," Nos. 1, 2.

[3] It should be remarked that the symptoms here described (in accordance with the opinion of most authorities) as due to alcohol, are believed by some to be met with only in drinkers who are smokers, and to be really due to tobacco. See Nettleship, "St. Thomas's Hosp. Rep.," 1879. If the fact is true that smokers who drink suffer less from tobacco amblyopia than do abstainers from alcohol, additional doubt is thereby cast on the influence of the latter in causing the same symptoms.

Galezowski as a symptom of retinal anæsthesia, in some cases of chronic alcoholism. It is probable, indeed, that the perception of green is commonly lost as well as that of red, both colours appearing grey.[1]

A stage of congestion, before the onset of the atrophy, has been described by Allbutt and others. The appearances were generally those already described as "Simple Congestion" (p. 44), uniform redness of the disc, with softened edges. The disc gradually becomes paler, and ultimately passes into white or greyish atrophy, often with small vessels. Uhthoff[2] examined a thousand cases of severe alcoholism in inmates of asylums, and found that 13·9 of these suffered from pathological whiteness of the temporal half of each disc, with a central scotoma in every case. He found this condition in only one out of a hundred apparently healthy men, whom he selected for comparison. Moreover, Moeli[3] has stated that he has detected changes in the optic disc in 15 per cent. of the cases of delirium tremens examined by him. When the condition of the nerve has been ascertained by microscopical examination, granular degeneration of the nerve fibres has been found in some cases. Out of seven cases examined post-mortem by Uhthoff, two showed distinct interstitial neuritis, with marked increase of the connective tissue. The changes were most distinct just behind the globe, and did not extend far back. I believe that congestion, sometimes with slight œdema, is occasionally to be recognized in the cases of chronic alcoholism. It is, no doubt, an analogous condition to that change in the meninges which leaves the thickening and opacity often to be found after death.

The progress of the atrophy is slow, and the prognosis better than in many other forms; considerable good being effected, especially in the pre-atrophic stage of amblyopia, by strychnia and tonics. Complete recovery of vision may take place, although the pallor of the disc continues.[4]

[1] Nuel: "Ann. d'Oculistique," Sept. 1878.
[2] "Ophth. Rev.," vii. p. 100.
[3] "Neurol. Centralbl.," 1884, p. 260.
[4] Berry: "Ophth. Rev.," iii. 1884, p. 101.

In a fatal case of alcoholism, Lawford found during life widespread cloudiness of the retina, with normal discs, and without any central colour scotoma. After death the retina of one eye was examined by Edmunds[1] and himself; there was œdema of the nerve-fibre and ganglion-cell layers, and in the outer nuclear layer there were spaces, filled with a clear effusion, between the Müllerian fibres. In a case of severe alcoholic paralysis related by Ord,[2] well-marked double retinitis was found, with white patches.

In acute alcoholism, ophthalmoscopic changes are not commonly present. In one case Jäger found a condition of diffuse retinitis, with numerous hæmorrhages, in a patient suffering from delirium tremens. This was undoubtedly an exceptional complication, but we need more facts regarding slight changes.

Tobacco Poisoning.

The occurrence of defective sight from tobacco smoking was described in 1854 by Mackenzie, who was inclined to attribute most cases of amaurosis to this cause. The subject attracted little attention until Hutchinson,[3] in 1864, brought forward facts to show that amblyopia, accompanied by slight ophthalmoscopic changes, often results from this cause, while Förster[4] and Hirschberg[5] have demonstrated that the affection of sight uniformly presents special characteristics. The relation between these symptoms and tobacco smoking has indeed been doubted by some writers, but must be regarded as now among the best established facts of ophthalmology.[6]

Förster has remarked that the sight suffers from tobacco

[1] "Trans. Ophth. Soc.," ix. 1889, p. 137.

[2] "Lancet," Feb. 11, 1888; see also Sharkey, "Trans. Path. Soc. of Lond.," 1889, p. 359.

[3] "Lond. Hosp. Rep.," 1864; see also "Med.-Chir. Trans.," 1867; "Ophth. Hosp. Rep.," 1871 and 1876.

[4] Graefe u. Saemisch's "Handbuch," vol. vii. p. 201.

[5] "Deutsche Zeitschrift f. Prakt. Med.," 1878.

[6] For an excellent *résumé* of what is known on the subject, see Nettleship's "Notes on the Diagnosis of Tobacco Amblyopia," "St. Thomas's Hosp. Rep." 1879.

generally between thirty-five and sixty-five years of age, and believes that tobacco is not so well borne during the second half of life as during the period of full vigour. Several of Hutchinson's cases, however, were under thirty. Mental trouble, with its accompanying sleeplessness and loss of appetite, seems often to be the determining cause of the poisoning in men long habituated to the use of tobacco, and that even where there is no history of a recent increased indulgence. It has been thought that the disease occurs more frequently among abstainers from alcohol than among those who take alcohol, and some facts mentioned by Nelson[1] seem to show that in the latter the affection may come on more slowly.

The failure of sight is gradual and usually equal in both eyes, unaccompanied, as a rule, with headache or other cerebral symptoms. It is nearly always more marked in a bright than in a dull light. The characteristic of the failure is the presence of a defect in the centre of the field of vision, a "central scotoma," transversely oval or oblong, extending from the fixing point to the blind spot, and often embracing both. It is a relative, not an absolute scotoma; there is dimness, not loss, of sight, and the failure is greater for certain colours (green and red) than for white. If the defect is slight, the coloured object must be of small size in order to detect it. The scotomata are symmetrical, or nearly so, in the two eyes (see Figs. 60, 61, p. 126), and seem to begin most commonly at or near the fixing point (Leber, Treitel, Nettleship). Nelson, however, has described a case in which the scotoma surrounded the blind spot and the fixing point was free. The variations in the exact limits of colour fields in different individuals and with different degrees of illumination render it uncertain whether there is a peripheral limitation of these fields; such limitation is certainly not always present, but probably may be in severe cases (Treitel, see also Fig. 59, p. 126). The symmetry of the scotomata is anatomical, not functional, and indicates a morbid process in the orbital portions of the optic nerve, doubtless in its axis

[1] "Brit. Med. Jour.," 1880, ii. p. 774.

(see p. 123). Sometimes a stage of simple congestion, a "hazy disc," slight uniform redness, with soft edges, without noticeable swelling, may be the first change. In exceptional instances, slight papillitis, with a few small retinal hæmorrhages near the disc, has been found; in these cases the failure of sight has been unusually rapid, and there has been a history of recent great excess in tobacco. Later there is a slight degree of atrophy.

The treatment consists essentially in the removal of the cause. Tonics and hypodermic injections of strychnine are also of use, especially in the pre-atrophic stage. Hutchinson believes the prognosis in most cases to be good, three-fourths of his cases having recovered, or presented great improvement in sight. Age does not render the prognosis worse.[1]

Quinine.

Quinine in large doses may cause complete temporary amaurosis. Many well-marked cases have been recorded.[2] The amount of quinine which caused the symptoms varied from a minimum of 80 grains in thirty hours, to a maximum of 1,300 grains in three days. In most of these cases the quinine was given for malaria, but that the affection of sight was due to the former, and not to the latter, is proved by the definite and peculiar character of the symptoms, and by the fact that, in some other cases, the patient was not suffering from any disease, and the quinine was taken by accident. In all the cases the loss of sight was at first complete, and was associated with loss of hearing. The deafness soon passed away, usually in twenty-four hours. The blindness con-

[1] For much valuable information on this subject, the reader is referred to the "Report on Toxic Amblyopia," "Trans. Ophth. Soc.," vol. vii. 1887, p. 85.

[2] Giacomini: "Ann. Univers. di Med.," 1841; Graefe: "Arch. f. Ophth.," iii. pt. 2, p. 396; Roosa: "Archives of Ophthalmology," vol. iii. p. 3, and ix. pt. 1; Gruening: ibid. vol. x. pt. 1, p. 81; Vorhies: "Trans. American Med. Assoc.," 1879; Michel: "Archives of Ophthalmology," x. pt. 1, p. 102; and Knapp: ibid. x. pt. 2, p. 220. The last paper contains a very full discussion of the subject. See also papers by Browne, "Trans. Ophth. Soc.," vol. vii. p. 193 (with references to previously recorded cases), and Nettleship, in same volume, pp. 218, 219.

tinued for a longer time, which varied according to the dose. Central vision returned to the normal in a few days, weeks, or months, but the peripheral vision continued lost for a very long time. This contraction of the visual field after the return of central vision seems to be invariable, and the restricted field is usually transversely oval. Colour-vision is also impaired. The pupils are dilated, and during total blindness are irresponsive to light, but act to accommodation (Gruening). The ophthalmoscope has shown pallor of the disc, and in all cases a remarkable diminution in size of the retinal vessels, which may be reduced to threads, and emptied by the slightest pressure on the eye. A cherry-red spot at the macula has been noticed (Gruening). Vorhies found the choroidal vessels also empty. In the case of Giacomini, where three drachms were taken at a single dose, there was loss of consciousness at the onset. In all cases a considerable degree of recovery has ultimately occurred. In the most severe case (Michel), in which seven drachms of quinine were taken, there was no improvement for several months, and it was thought that sight was permanently lost; nevertheless, fifteen months afterwards acuity of vision was nearly normal, although the fields were much restricted. The vessels had increased in size, but were still much below the normal. Recovery in six weeks has followed a dose of five drachms. Whilst the symptoms are passing off, relapses may be produced by insignificant doses of quinine.

Bisulphide of Carbon.

Bisulphide of carbon was the apparent cause of a "peri-neuritis," ending in partial atrophy, in a case recorded by Galezowski.[1] Atrophy of the optic nerves is also seen, not very rarely, among the workers in india-rubber factories, in which bisulphide of carbon is used. A special committee of the Ophthalmological Society (consisting of Messrs. Frost, Gunn, and Nettleship) was appointed to investigate this form of toxic amblyopia, and in their very valuable

1 Galezowski: "Des Amblyopies et Amauroses Toxiques," p. 141.

report[1] on the whole subject they tabulate twenty-four cases of amblyopia coming on after the development of other symptoms indicating great depression of the whole nervous system. In most cases examined there was a distinct central colour-scotoma, and the ophthalmoscope showed pallor and blurring of the edge of the optic disc, with loss of transparency of the retina for some distance from the disc.

Other Poisons.

Silver poisoning is said to be accompanied by amblyopia, in addition to the other symptoms of argyria. No ophthalmoscopic changes have, however, been recorded, but silver has been found in the eyeball (sclerotic sheath of the optic nerve, &c.), by Reimer, deposited in small round granules. The effect of silver is closely analogous to that of lead. It may, as I have seen, lead to wrist-drop, gout, and albuminuria, and it is therefore highly probable that the same ocular changes may, in some cases, result.

In *mercurial poisoning* amblyopia has been observed; in one case optic neuritis existed,[2] and in another optic nerve atrophy.[3] Of ocular changes in copper and phosphorus poisoning nothing is known.

Salicylic acid may cause amblyopia, but without changes in the fundus oculi. The same effect has been observed from salicylate of soda.[4]

ACUTE GENERAL DISEASES.

Typhus Fever.

Loss of sight has been many times observed during convalescence from typhus fever,[5] and subsequently atrophy of

[1] "Trans. Ophth. Soc.," vol. v. 1885, p. 157.
[2] Square: "Ophth. Hosp. Rep.," vi. p. 54.
[3] Galezowski: "Des Amblyopies et Amauroses Toxiques, p. 141.
[4] Gatli: "Gaz. degl. Ospital," 1880, i. 4.
[5] In a considerable number of the cases recorded abroad it is doubtful whether the disease was typhus or typhoid fever. The cases on which the statements in the text are founded appear to have been true typhus.

one or both optic nerves has been found. In some of these cases there have also been cerebral symptoms, as in a case recorded by Benedikt, in which left hemiplegia was accompanied by atrophy of the right optic nerve. In such cases, probably, the atrophy was the result of a cerebral lesion. In other cases there were no symptoms except those in the eye, and a primary affection of the optic nerve appeared to have occurred. In some cases these ophthalmoscopic changes have been those of simple atrophy, but in others, where the affection of sight was first noticed during convalescence, optic neuritis has been found.[1] In a case at Great Ormond Street Hospital, marked papillitis was found by Penrose and Gunn during the height of the fever. Of the origin of the neuritis nothing is known.

Typhoid Fever.

The occurrence of amblyopia and amaurosis during convalescence from typhoid is well established,[2] although rare. It may or may not be attended with ophthalmoscopic changes. In the latter case the prognosis is favourable; the affection usually passes away in the course of two to eight weeks. The form of amblyopia varies; anæsthesia of the retina has been observed by Leber, and an annular defect in the field by Hersing.

When ophthalmoscopic changes have been observed, there has been simple atrophy, single or double, without preceding inflammation; or double neuritis may be present, ending in atrophy, partial or complete, or less commonly in recovery. Hutchinson has, for instance, recorded[3] the case of a boy whose sight failed at three years and a half, two to four weeks after a fever with diarrhœa and headache, a sister having suffered from similar symptoms at the same time. Symmetrical neuritis was found. and ten years later white

[1] Teale: "Med. Times and Gazette," May 11, 1867. Chisholm: "Ophth. Hosp. Rep.," vol. vi. p. 214.

[2] Nothnagel: "Deut. Arch. für Kl. Med.," 1872, ix. p. 480.

[3] "Ophth. Hosp. Rep.," ix. p. 125.

atrophy with small vessels. The neuritis is so rare that Leber suggests, as Stellwag v. Carion had suggested long before,[1] that the cases in which it is found may really have been cases of meningitis which have been mistaken for typhoid fever—an error not very rare. It must be remembered, however, that neuritis does occasionally follow other acute specific diseases. It has been thought that the cases accompanied by hyperæmia of the discs are cases complicated by meningitis; but meningitis, except as secondary to suppuration in the ear, is exceedingly rare in typhoid fever. Sir William Jenner has informed me that he has never seen it. It does not appear from Dr. Murchison's work on Fevers, that he had ever met with a case. To infer meningitis in consequence of extreme delirium or coma is certainly not warranted by pathological facts.

Extreme narrowing of the retinal arteries, on both sides, with pallor of the disc and loss of sight, was found by Heddaens[2] in a case of great emaciation after typhoid. On good food the arteries regained their normal size, but the disc remained pale, and sight did not improve beyond $\frac{1}{20}$.

Galezowski[3] and Snell[4] have observed embolism of the central artery of the retina during convalescence from typhoid.

Relapsing Fever.

It is well known that extensive intra-ocular inflammation is apt to follow relapsing fever. Trompetter[5] found it in 21 out of 325 cases, or six per cent. There was inflammation of the choroid and ciliary body with hypopyon, but without iritis. There were also opacities in the vitreous, amblyopia, and limitation of the field. Its origin is doubtful. Throm-

[1] "Ophthalmologie," Bd. ii. Abt. I. 1855, p. 662.
[2] "Monatsbl. für Augenheilk.," Aug. 1865.
[3] "Traité Iconographique," p. 188.
[4] "Ophth. Rev.," i. p. 403.
[5] "Klin. Monatsbl.," April, 1880, p. 123.

bosis in vessels or embolism from the spleen has been assumed as its cause (Blessig, quoted by Trompetter).

Measles.

Amblyopia, without ophthalmoscopic changes and ultimately improving to the normal, has been seen, as a sequel to measles, by v. Graefe and Nagel; in some cases accompanied by cerebral symptoms, convulsions, and sopor. Nagel has also met with three cases of optic neuritis after measles, but in the epidemic in which they occurred there were many cases of meningitis. In three other cases lately recorded by Wadsworth [1] there were also symptoms of meningitis. Dr. Stephenson [2] has recently reported a well-marked case of optic neuritis after measles, without any symptoms indicative of meningitis. The observation is valuable, since the eyes were examined shortly before the attack, and the fundi found normal. The discs have become paler than formerly, Dr. Stephenson writes, but are not completely atrophied, and there is still good vision. As Förster remarks, the commonness of the disease, and the rarity of affections of sight in it, show that the connection between the two cannot be a very close one.

Scarlet Fever.

The frequency with which renal disease accompanies and succeeds scarlet fever renders affections of sight not very rare consequences of the disease. Occasionally, however, they arise independently of any renal disturbance.

Uræmic amaurosis is common in scarlatinal dropsy. It comes on suddenly, when the renal disease is at its height, is commonly complete, double, unattended by ophthalmoscopic changes, and passes away. Occasionally, cerebral symptoms accompany it—convulsions, and, in rare cases, hemiplegia, from a cerebral thrombosis or embolism, which persists after the cessation of the convulsions, and the return of sight.

1 "Boston Med. and Surg. Journal," vol. ciii. p. 636.

2 "Trans. Ophth. Soc.," vol. viii. 1888, p. 250.

Neuro-retinitis has, however, been observed to succeed scarlet fever when there has been no renal disease or albumen in the urine. Betke[1] has recorded a case in which there was great dimness of sight seventeen days after desquamation. There was no albuminuria, but a marked neuro-retinitis was found on ophthalmoscopic examination, less developed in the right eye than in the left. There was no sign of meningitis, past or present. The neuritis entirely disappeared, and sight was restored in eight weeks. A similar case has been recorded by Pflüger.[2] A child, ten years old, became blind three weeks after an attack of scarlet fever, the loss of sight being complete at the end of three or four days. During the fever there had been considerable headache. When sight was lost, double papillo-retinitis was found to exist. The arteries were narrow and tortuous, with slight pulsation; the veins were dilated; there was considerable swelling, and some hæmorrhages existed. A month later sight had much improved, but four months after it was not quite normal, and the neuritis had not entirely subsided. The urine throughout was free from albumen. In a case recorded by Hodges[3] the loss of vision was due to embolism or thrombosis of one retinal artery. The amaurosis was complete and permanent in the affected eye.

It is not uncommon to meet with atrophy of the optic nerve after scarlet fever, and the atrophy may have all the aspects of a consecutive atrophy. It has been observed in association with the symptoms of a local cerebral lesion, hemiplegia, &c. (Loet), but in some cases has occurred alone. Two remarkable cases have been recorded by Bayley,[4] in which, in two sisters, sight gradually failed some months after an attack of scarlet fever, without albuminuria or dropsy. One became blind and idiotic, and the other epileptic. The tint of the optic discs was "pale but not the bluish-white of atrophy," and the fundus in each case showed accumulation of pigment.

[1] "Monatsbl. für Augenheilkunde," Bd. viii. 1869, p. 201.
[2] "Arch. f. Ophth.," xxiv., pt. 2, p. 180.
[3] "Ophth. Rev.," iv. p. 296.
[4] "Lancet," Sept. 15, 1877.

It must be remembered that an intense albuminuric inflammation may leave partial atrophy of the optic nerve.

Variola.

Leber has observed diffuse neuro-retinitis in variola during the stage of drying of the eruption. In a case which came under my own observation, atrophy of one optic nerve appeared to have succeeded small-pox. (See Case 60 in previous editions.)

Acute Rheumatism.

Acute rheumatism is not usually associated with any changes in the fundus oculi. Embolism of the cerebral arteries sometimes, though rarely, occurs during the course of an attack, but embolism of the retinal arteries has not, I believe, been observed except as a late sequel of the resulting endocarditis. Schmidt once observed irido-choroiditis (such as is common in relapsing fever) after an attack of acute articular rheumatism without endocardial complication.[1]

Malarial Fevers.

Changes in the fundus oculi are present in some cases of malarial fever; rarely in the intermittent of this country, but not uncommonly in the severer forms of malarial fever, especially in tropical climates. Poncet,[2] for instance, found changes in ten per cent. of the cases of malarial cachexia in Algeria. The changes which have been observed consist of retinal hæmorrhages, neuro-retinitis, and atrophy of the optic nerve.

Hæmorrhages may occur without other change, sometimes in the posterior segment of the eyeball, sometimes chiefly in the ciliary region (Poncet). Three instances of retinal hæmorrhages in ague have been recorded by Stephen Mackenzie.[3] One was a young man, aged twenty, who had

[1] "Arch. f. Ophth.," Bd. xviii.

[2] "Ann. d'Oculistique," May, 1878.

[3] In a paper on "Retinal Hæmorrhages and Melanæmia as Symptoms of Ague," "Med. Times and Gaz.," 1877. I am much indebted to Dr Mackenzie for the woodcuts from his paper.

one attack of ague on his way home from India, and a severe paroxysm immediately after his arrival. The attacks recurred daily for a fortnight, when he came under treatment, and numerous retinal hæmorrhages were found, most numerous near the disc, chiefly along the course of the larger vessels, especially arteries, which they in places obscured (Fig. 80). Sprinkled about the fundus, and most numerous near the disc, were many small round bright spots, resembling pin-holes pricked in a piece of paper held up against the light.

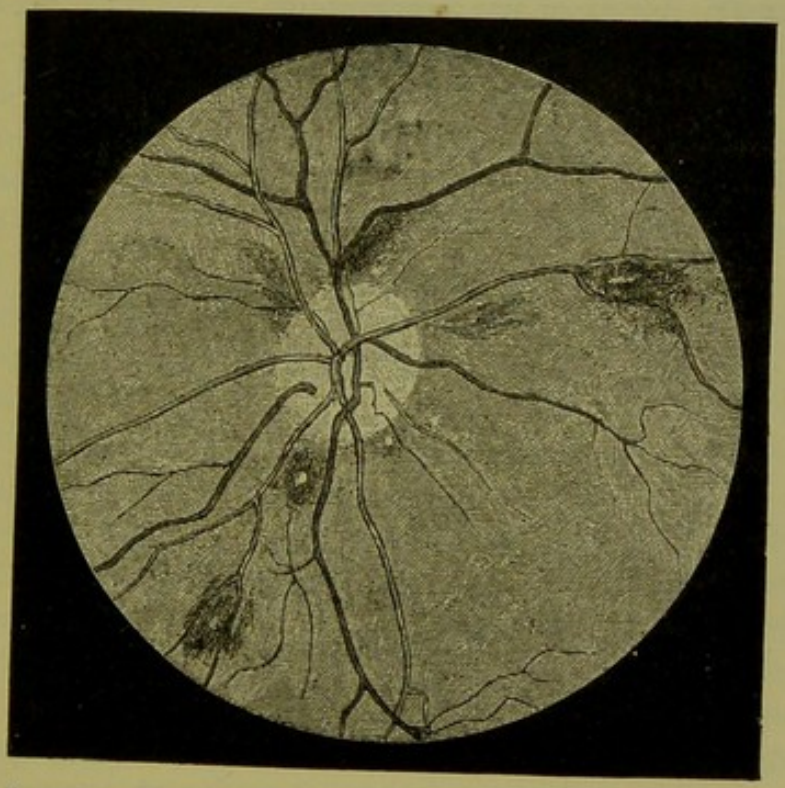


FIG. 80.—RETINAL HÆMORRHAGES IN AGUE (MACKENZIE).

The retinal vessels were of normal size, and their sheaths did not appear thickened. These hæmorrhages were carefully observed day by day, and were seen to fade away gradually; and, as each died away, it left, to mark its former situation, one of the shiny white spots of which mention has been made above. There was no albuminuria or other symptom of Bright's disease. The spleen was large. The blood at first contained much pigment, but after the first few days no more could be found.

In two cases, at the Seamen's Hospital—a man, aged twenty-nine, with quotidian ague, and another, aged eighteen, with tertian ague—hæmorrhages were found; in the former case, numerous, large, and superficial, leaving white patches. One was paler in the centre than in the periphery (Fig. 81). They quickly disappeared. Neither of these patients had melanæmia. In several cases subsequently examined, no hæmorrhages were found.

Hæmorrhagic retinitis has also been met with by von

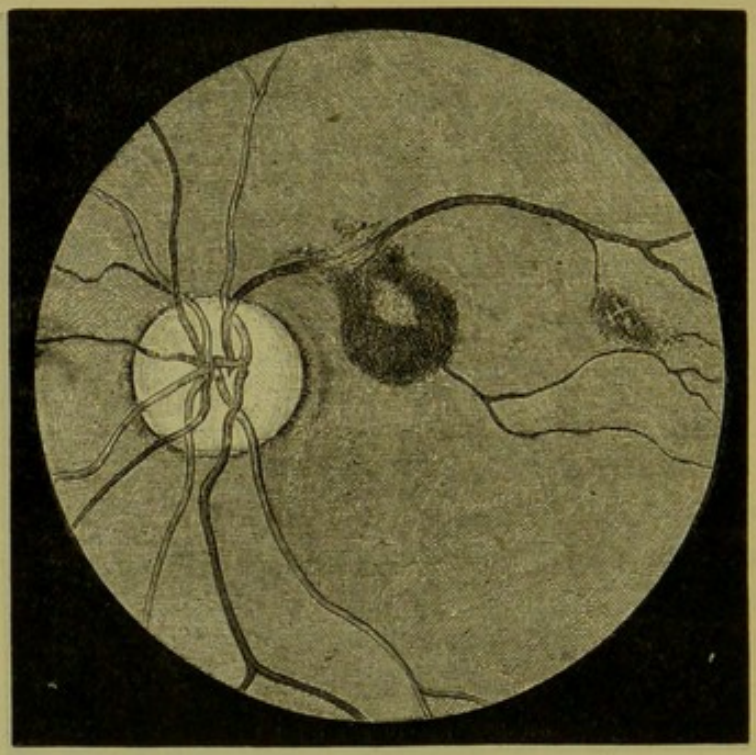


Fig. 81.—Retinal Hæmorrhages in Ague (Mackenzie).

Kries.[1] One patient, who had suffered from ague for a week only, had an extensive hæmorrhage into the vitreous. The intermission had been arrested by quinine, and the first freedom on the day of periodical recurrence was accompanied by the hæmorrhage.

Poncet observed, in Algeria, besides hæmorrhages, peri-papillary œdema and even considerable neuro-retinitis. He also found, in the retinal and choroidal vessels, large cells

[1] Arch. f. Ophth.," vol. xxiv. pt. 1, p. 159.

containing leucocytes and pigment. Neuritis has also been seen in one case by Galezowski,[1] and in two by Hammond,[2] unilateral, with stellate deposits of pigment in the retina following the course of the vessels.

Atrophy of the optic nerve has also been observed to succeed malarial fever. It is very rare, however, as a consequence of the intermittents of temperate climates, although a few cases are on record. After the severe malarial fever of hot climates it is not infrequent. Several cases are narrated by Galezowski [3] and by Bull.[4] The disc is white, the vessels are small, and the field of vision is greatly restricted. The pathology of the retinal changes is still obscure. The retinal hæmorrhages have been ascribed to pigmentary embolism, but they are, as Mackenzie has shown, to be found when there is no melanæmia. Poncet attributes them to the blockade of minute vessels by leucocytes. The atrophy was ascribed by Galezowski to pigmentary embolism. It seems possible that the atrophy may be the result of such neuro-retinitis as is described above, and which, damaging sight only during the stage of atrophy, attracted no attention during its acute stage.

Two remarkable cases which have been recorded by Ramorius,[5] suggest that spasm of the retinal vessels may be a consequence of malarial poisoning. The chief symptom was periodical amblyopia, and during one of the attacks the optic discs were pale, the retinal arteries were filiform and almost bloodless, and the veins were scarcely perceptible. At the same time there was great congestion of the face and ears, and a sensation of heaviness in the head. Each attack was attended with a sensation of coloured circles moving from the periphery of the field towards the centre. In the intervals between the paroxysms the appearance of the fundus oculi was normal. Bromide of potassium had no effect, but quinine quickly cured each case.

[1] "Traité Iconographique," p. 190.

[2] "Trans. American Neurological Society," 1875.

[3] Loc. cit.

[4] "American Journ. of Med. Science," 1877, p. 403.

[5] "Annali di Ottalmologia," 1877, pt 1, and "Ann. d'Oculist.," vol. lxxxii. p. 200.

Purulent affections of the eye (choroiditis,[1] iritis, &c.) such as are seen in pyæmia, have been described in intermittent fever, but are extremely rare, and some doubt may be felt regarding the diagnosis of the original disease when it is remembered how closely some cases of pyæmia simulate intermittent fever. Even the influence of quinine, on which diagnostic weight is often laid, is not entirely conclusive.[2]

Erysipelas.

Erysipelas of the face is sometimes followed by loss of sight and by the signs of atrophy of the optic nerve (v. Graefe, H. Pagenstecher, Hutchinson, and others). It is produced by the extension of the cellulitis into the orbit, and the resulting damage to the trunk of the optic nerve by invasion or pressure. V. Graefe has pointed out that there is commonly some exophthalmos, but this may be very slight, and may bear no proportion to the subsequent damage to sight. In most recorded cases any symptoms suggestive of orbital cellulitis have escaped notice, probably from the difficulty of the examination. In one, however (that of Story[3]), there was permanent limitation of the ocular movements. The loss of sight often comes on rapidly. In one of Pagenstecher's cases amaurosis was complete at the end of fourteen days. Early observations of neuritis or neuro-retinitis have been recorded by Vossius and Lubinski,[4] and slight opacity of the retina has been seen by many observers. It rapidly passed into atrophy. Usually, however, as soon as the examination could be made, there has been pallor of the disc and remarkable narrowing of the vessels, the arteries especially. Jäger has recorded, for instance, a case in which an adhesion of the eyelids

[1] Peunoff: "Centralbl. für Augenk.," 1879, p. 120.

[2] For example, in a case of this kind described by Landesberg, in which, although quinine cut short the affection, abscesses formed during convalescence, in one toe and the forearm.

[3] "Brit. Med. Journal," March 16, 1878.

[4] Lubinski: "Klin. Monatsbl.," April, 1878, p. 168; Pflüger of Berne: "Augenklinik Bericht" for 1877; and Virchow's "Jahresbericht," 1878, vol. ii. p. 438.

required division with the knife five weeks after the erysipelas; the optic disc was grey and atrophied; one branch of the central artery and its corresponding vein were normal, the others reduced to lines with white borders. In Story's case some arteries were bloodless, and occluded veins were represented by dark radiating lines.

It is probable that thrombosis in the central artery is not infrequently the mechanism by which the effect is produced. Thus, August[1] found the ophthalmoscopic appearances similar to those in embolism (arteries either invisible, or, in places, transformed into white lines), in a case in which the erysipelas caused orbital cellulitis, and in addition visible clotting in supra-orbital and frontal vessels; he believes that the organism penetrates the walls and causes inflammation and clotting. Knapp,[2] however, urges that the mechanism is compression of vessels in the orbit. In an early case he found the veins distended with stagnant blood. He quotes Panas, who found obliteration of the retinal artery. In a case observed by Nettleship, although the arteries were small they pulsated on pressure. It seems to me probable that the mechanism in these cases is not always the same.

In one of Pagenstecher's cases there was a central scotoma and also peripheral limitation of the field. Necrosis of the nerve, less complete at the lamina cribrosa than farther back, was found by Nettleship.[3] Opacity of the vitreous and glaucoma have also been seen after erysipelas.

Diphtheria.

The defect of sight which so often follows diphtheria, and is due to a paralysis of accommodation, is not attended with any ophthalmoscopic change. In rare cases, however, vision is defective, apart from the paralysis of accommodation, and in such cases one or two observers (*e.g.* Bouchut) have found congestion of the disc, simple or with œdema sufficient to veil the edges and even, in part, the vessels, and in very rare

[1] "Klin. Monatsbl.," 1884, 43.
[2] "Arch. f. Augenkr.," 1884, i. 83.
[3] "Trans. Path. Soc.," vol. xxxi. 1880, p. 254.

cases an actual neuritis which may go on to atrophy. The atrophy may be unilateral, as in one case figured by Bouchut. This case, however, was accompanied with partial right hemiplegia and defect of speech. The congestion and œdema are usually bilateral, but may be more intense on one side than on the other. I have also seen one definite case of primary atrophy after diphtheria. The patient was a woman, aged forty-one, with a family history of epilepsy. Shortly after the diphtheria she suffered from numbness of the arms, from paralysis of the palate and of accommodation, and from double vision. With the exception of the diplopia these symptoms passed off; but, a little later, slight weakness of the right side developed and became permanent. Along with this there occurred progressive failure of sight, and two years later there was well-marked primary atrophy of the optic nerves, with considerable amblyopia. The pupils did not react to light in the least, and but slightly to accommodation. There was nystagmus on looking to the left, and the upward movement of the eyes was completely lost. There were no other signs of tabes, and the knee-jerks were perfect.

Parotitis.

Transient dimness of sight may succeed mumps, and a coincident congestion of the optic nerve has been described by Hating.

Tonsillitis.

In a case of tonsillitis v. Graefe once saw signs of diminished blood-supply to the retina accompanying sudden loss of sight. The known relation of tonsillitis to rheumatism suggests the probability of embolism in this singular case.

Whooping-Cough.

Blindness has been observed to come on during the progress of whooping-cough, and in one case Knapp[1] found the discs white, and the retinal arteries invisible in one eye and mere

1 "Arch. of Ophthalm. and Otol.," vol. iv. Nos. 3 and 4, p. 448.

lines in the other. The patient was very weak, and Knapp suggests as explanations, anæmia from cardiac weakness, or hæmorrhage into the nerve-sheaths. Landesberg[1] also observed in one case symptoms of partial embolism, serous infiltration into the retina, slight swelling of the papilla, a red macula, thin arteries, engorged and tortuous veins. Two upper arterial branches were found to be permanently obstructed. In another case he observed ecchymoses in the retina. According to Loomis (quoted by Knapp), loss of sight generally occurs, in this disease, in children who are much prostrated, and who commonly die from lobular pneumonia.

Cholera.

In cholera v. Graefe found that, during the state of collapse and cyanosis, the circulation in the smaller, and even in the middle-sized, arteries may apparently cease. When the weakness of the heart was moderate, the artery pulsated on slight pressure with the finger on the eyeball; but when the heart was strong this could not be well produced. If the heart was so weak that the radial pulse could not be felt, and the second sound of the heart was inaudible, slight pressure on the eye caused emptying of the arteries without pulsation. The veins were large and dark, visible in the finest divisions. The papilla was of a pale lilac tint.

Pyæmia and Septicæmia.

The occurrence of a general inflammation of the eye in cases of septicæmia of various kinds, "metastatic panophthalmitis," has long been known, but it is only during the last few years that the use of the ophthalmoscope in medical and surgical practice has revealed the fact that slighter retinal changes are present in a large proportion of the severer forms of these affections, and constitute a symptom of considerable diagnostic and prognostic importance, as well as of great pathological interest. The knowledge of their character is

[1] "Med. and Surg. Reporter," Sept. 8, 1880.

largely due to the labours of Heiberg,[1] Roth,[2] and especially of Litten.[3] All forms of affection are most common in the intense septicæmia of puerperal women, but are also met with in other cases.

Panophthalmitis.—The general inflammation of the eye, "pyæmic or metastatic ophthalmia," is usually attended with suppuration in the various structures—iris, choroid, retina, vitreous—with rapid destruction of the eyeball. It was shown by Virchow[4] to depend upon septic embolism, and later researches have fully confirmed the fact. Plugs in the vessels have been found by Virchow, Roth, and Heiberg. The latter found micrococci in the emboli. It is usually associated with the endocarditis which is so common in septicæmia. Virchow found yellowish granular masses in the capillaries of the retina, similar to those which were present in the cardiac valves, and he, with most subsequent observers, regarded the cardiac valves as the source of the emboli. The condition may, however, occur independently of any endocarditis.[5] Even in such cases, however, the presence of infarcts in other organs, and of suppurating thrombi in the source of the septicæmia, demonstrated the probability of embolism, although not directly from the heart. It is well known that pyæmic emboli may pass through the lungs and lodge in the general system. The septic inflammation excited in the eye may start from the choroid or the retina, as is demonstrated by two cases of Litten's, in which the process commenced a short time before death, and he found plugging in the one case of choroidal and in the other of retinal vessels. A case in which the mischief apparently commenced in the retina has also been described by Roth. When the retinal vessels are plugged, hæmorrhages in the retina are invariable, as Virchow demonstrated, and the commencement of the process

[1] "Med. Centralblatt," 1874, No. 36.

[2] "Deut. Zeitschrift für Chirurgie," 1872, p. 471; Nagel's "Jahresbericht," 1872, p. 349.

[3] "Charité Annalen" for 1876, p. 160.

[4] "Arch. f. Path. Anat.," Bd. x. 1856.

[5] Litten: loc. cit. Case 8; Meckel: "Charité Annalen," Bd. v.; Virchow: "Ges. Abhand.," p. 539; Schmidt: "Arch. f. Ophth.," xviii. p. 1.

in retinal hæmorrhages, with opacity of the retina and vitreous, may be watched with the ophthalmoscope.[1] The opacity of the retina depends apparently in most cases on acute degeneration. It was found by Roth to be merely softened, and containing granule cells, although the other structures of the eye were infiltrated with pus. A layer of pus has, however, been seen on the surface of the retina, and pus is said to have been found in some cases in the nerve-fibre layer. Rarely, the changes have been found limited to a small area of the retina and the adjacent choroid.

It is probable that this severe ocular inflammation is always produced by the agency of septic organisms circulating in the blood. These, in the form of bacterial or micrococcal masses, have been found in the vessels of the eye in many cases.[2]

The affection is usually single, but in many cases both eyes are affected, it may be unequally. It occurs only in intense forms of septicæmia, commonly not long before death. In rare cases it may occur when the general symptoms of the disease are not advanced, as in a case mentioned by Litten, in which a woman came to the hospital with one eye in a state of complete suppuration, but with so little subjective symptom of the considerable fever which was found to exist, that she was unwilling to remain. Death occurred some weeks after the eye was lost. Ophthalmoscopic examination of the sound eye revealed no change for some time after admission. One day choroiditis and infiltration of the vitreous was discovered; the same day rigors and joint-inflammation occurred, and in three days later the patient was dead.

Retinitis Septica.—Roth has described a peculiar form of retinitis in cases of pyæmia, characterized by the appearance of small white flecks in the neighbourhood of the papilla and macula lutea, varying in number, and occurring in most

[1] Litten: Case 8.

[2] Kahler: "Prag. Zeitsch. f. Heilkunde," 1879, iii., and "Centralbl. f. Med. Wiss.," 1880, p. 728; Pousson: "Arch. d'Ophth. Française," No. 2, Jan. 1881; Hosch: "Arch. f. Ophth.," vol. xxvi. pt. i, p. 177.

cases in both eyes. Sometimes small hæmorrhages were present. The white spots were found to consist of groups of swollen nerve-fibres, among which were granule cells, fattily-degenerated capillaries, and pigment granules. The affected spots were of small size, and showed little tendency to extension, or to the involvement of the vitreous or choroid. In no case observed by Roth was any plugging of vessels discovered, or any deposits on the cardiac valves, and he therefore believes that the change is due to the chemical alteration of the blood, but Kahler found micrococcal plugs. The affection was met with especially in cases in which decomposition was occurring in inflamed parts, such as extensive sloughing with secondary suppuration, and especially in pronounced septicæmia. It was found also in one case of putrid bronchitis.

Retinal hæmorrhages constitute, however, by far the most common and most important change in the fundus in cases of septicæmia. They usually accompany the suppurative panophthalmitis, especially when the process commences in the retina. They may also occur in the form described by Roth. But they may exist alone, without any sign of retinal inflammation, and as such constitute the most common ophthalmoscopic change in these cases. They have been very carefully studied by Litten, in cases of puerperal septicæmia, in which they almost invariably occur during the last two or three days of life. They are always bilateral, round, or irregular in form, and of variable size, sometimes very large. They are commonly adjacent to vessels, especially veins, but occasionally are situated apart from visible vessels. Most of the round extravasations present pale or white centres, which are often distinct as soon as the hæmorrhage appears. They are recognized without difficulty, some being always in the posterior portion of the fundus.

In some of the cases in which these hæmorrhages were seen, there was endocarditis, but in several cases recorded by Roth and Litten the heart was healthy. There is thus no necessary connection between the cardiac and the ocular condition. Moreover, the retinal change appears comparatively innocent;

no adjacent inflammation is excited. In no case could Litten find any plugging of the retinal vessels, and from these facts, he concludes with Roth, that embolism is not the cause of these extravasations, but that they are to be ascribed to the chemical change in the blood. This view is also supported by a case described by Leube,[1] but the same observer has recorded another case of septic pyæmia, secondary to

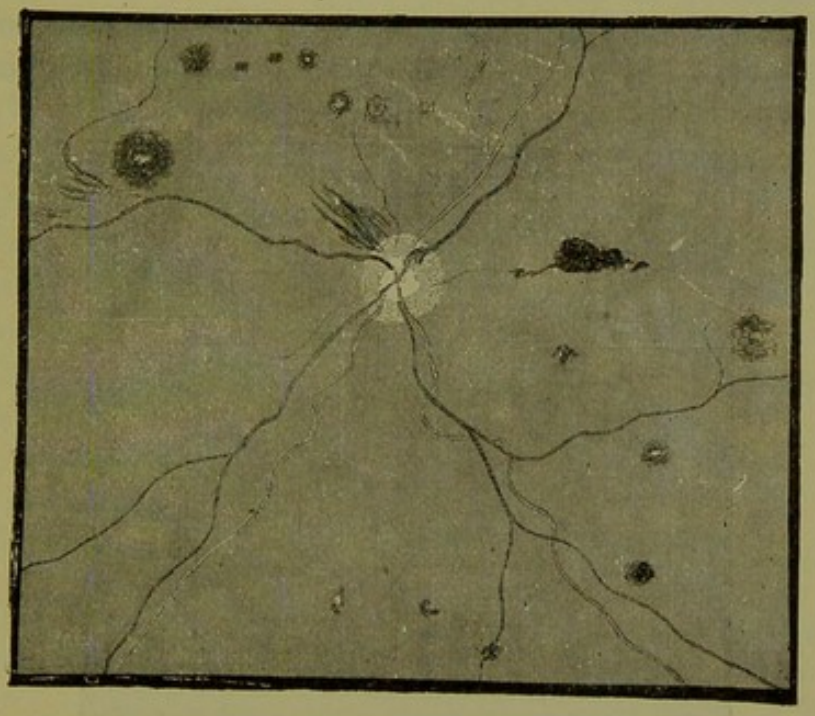


FIG. 82.—RETINAL HÆMORRHAGES IN A CASE OF ACUTE ULCERATIVE ENDOCARDITIS.

Some are striated in the nerve-fibre layer, others rounded in the deeper layers many have white spots in the centre.

double caseating epididymitis, in which retinal hæmorrhages existed, and, post-mortem, bacterial plugs were found in many other organs. The retinæ apparently were not examined. Rosenbach,[2] however, found a similar condition of multiple hæmorrhages in the retinæ of dogs, in which a septic endocarditis had resulted from experimental lesions of the valves, and he found micrococcal plugs in the retinal vessels

[1] "Deut. Arch. für Klin. Med.," xxii. 1878, p. 235.
[2] "Arch. für Exp. Path. und Pharmak.," 1878.

after death. From these facts we may conclude that, although simple hæmorrhages usually arise independently of embolism, they may sometimes be due to the plugging of vessels. In connection with the remark, that adjacent inflammation is often not excited, it may be noted that of six cases with endocarditis observed by Litten, in only three did the cardiac change present the aspect of malignant ulcerating endocarditis; in the other three the valves presented only innocent-looking vegetations. It is probable that the endocarditis varies in its degree of septic character in different

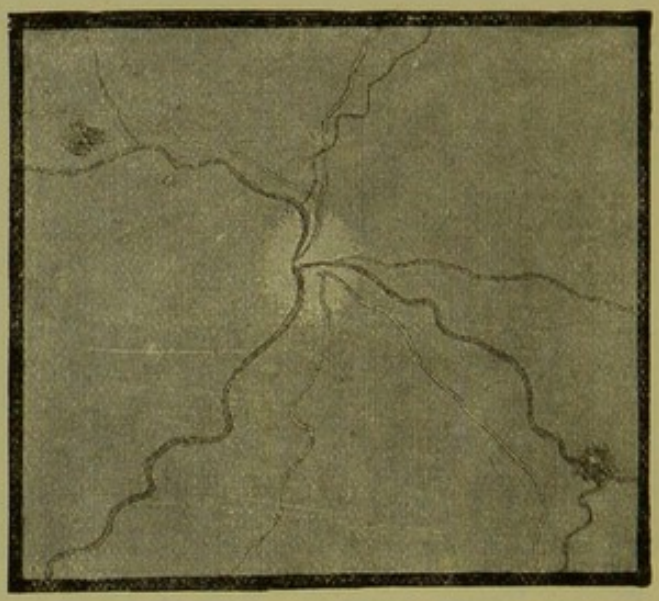


FIG. 83.—RETINAL HÆMORRHAGES IN A CASE OF ACUTE ULCERATIVE ENDOCARDITIS AND CHOREA.

The rounded hæmorrhage at the lower part of the figure has a white centre.

cases of blood poisoning. In several cases of pyæmia similar hæmorrhages have been noted on the mucous membrane of the conjunctiva or mouth (Litten, Leube).

From the fact that the retinal hæmorrhages usually precede death by a few days only, they afford important and very grave prognostic information. Now and then they are useful also in diagnosis, since they are apparently not found in acute specific diseases, even in those severe cases in which cutaneous hæmorrhages are present. Litten mentions two

cases of women admitted with high fever, cutaneous extravasations, and cardiac murmurs. One had been recently confined. They had the aspect of cases of septicæmia rather than of typhoid, but the absence of retinal extravasations led to a diagnosis of typhoid fever, which, in each case, was confirmed by a post-mortem examination. I have seen one case in which the presence of retinal hæmorrhages was of considerable assistance in establishing the fact that a post-puerperal illness, supposed to be typhoid, was really septicæmia.

The effect of the retinal hæmorrhages on vision can rarely be ascertained with exactness, on account of the general state of the patients, but they appear to cause little impairment.

Purulent meningitis sometimes occurs in cases of septicæmia. In one such case, recorded by Leube,[1] there were retinal extravasations, but after death intense inflammation of the optic nerves adjacent to the inflamed membranes was found.

THE OPHTHALMOSCOPIC SIGNS OF DEATH.

The stoppage of the heart's action and the consequent arrest of the circulation of the blood, which constitute the chief events in the cessation of systemic life, lead to striking changes in the fundus oculi, changes which are among the most unequivocal signs of death. Attention was first called to them by Bouchut in 1863,[2] and they have since been studied by many observers, especially by Poncet,[3] Arlidge,[4] and Gayet.[5]

As the heart's action is failing, the arteries may be observed to diminish in size (Arlidge). On the cessation of its contractions, the diminution in their size becomes more marked. A few minutes after death the capillary redness of the disc disappears, and its surface becomes of papery whiteness, in

[1] "Deut. Arch. für Klin. Med.," Bd. xxii. 1878, p. 263.
[2] "Traité des Signes de la Mort," 1863.
[3] "Arch. Gén. de Méd.," 1870, p. 408.
[4] "West Riding Asylum Reports," i. 1871, p. 73.
[5] "Ann. d'Oculistique," t. lxxiii. 1875, p. 5.

which, however, the central cup, if present, may appear of still more brilliant whiteness. The arteries quickly cease to be recognizable upon the disc, appearing to commence at its edge. On the fundus they are at first distinct, and usually narrow but otherwise of normal appearance. The veins may present normal characters, or may, like the arteries, quickly become indistinct upon the disc, appearing to start from its edge. Commonly the columns of blood within them soon become interrupted and broken up into segments, which give the vessels a beaded appearance. The indistinctness of the arteries, which is due to their contraction emptying them of blood, quickly extends towards the periphery, and in the course of half an hour, sometimes in ten minutes, they are unrecognizable. The veins remain distinct, but in most cases the beaded appearance increases. The choroid, during the first few minutes, presents nearly its normal tint, but this quickly lessens in intensity, and the colour which is presented depends on the amount of pigmentation. In dark eyes it acquires a yellow-brown colour, in lightly pigmented eyes it gradually assumes a pale, reddish-yellowish, sometimes a greyish, tint. Commencing opacity of the retina may sometimes be distinguished, and may be accompanied by a red spot at the macula lutea (Gayet), due to its freedom from opacity, and similar to that seen in embolism of the central artery.

These appearances persist until, generally after five or six hours, the progressive opacity of the media prevents further observation.

APPENDIX.

HOW TO SKETCH THE FUNDUS OCULI.

Nothing gives dexterity in the use of the ophthalmoscope so quickly and so effectively as an attempt to draw what is seen, and nothing gives ability to recognize details with accuracy and perceive every feature presented, as a habit of drawing does. Yet ophthalmoscopic drawing is hardly ever practised. It is supposed to be difficult, but it is neither difficult nor does it need any ability or facility for ordinary drawing. The process is within the reach of every student, and it may be well therefore to describe the method which is most useful. It is indeed sufficiently simple as scarcely to need even descriptive instruction, but it may be well not to assume that it can be discovered by each student for himself.

A pencil drawing should be first made, and from that either a more perfect pencil drawing on any paper that has a grain; or, what is better, a coloured drawing, which requires, however, a skill a pencil drawing does not need.

The disc should be drawn from $\frac{3}{4}$ inch to 1 inch in vertical diameter. The fundus should be observed first by the indirect method, and the outline of the disc made on a piece of paper by a faint pencil line, and other simple pencil lines should indicate the position of the chief vessels that can be seen by this method, the veins being made darker than the arteries. This being done, the observer should continue and complete the drawing by the direct method. If he is drawing the patient's left eye, his pencil and materials should be on a small table to his right; if he is drawing the patient's right

eye, this may be on his left or immediately in front of him, he sitting to the side of the patient. To continue the drawing he must turn his paper upside down. This brings the disc, as drawn from the indirect image, into the position in which it appears in the direct image. Both arteries and veins must now be represented, as they are seen, with a double contour. After indicating more precisely the position and outline of the physiological cup, some one large vein should be selected, and its position at the edge of the cup and edge of the disc noted, compared with the first drawing, and, if necessary, corrected. Then its double contour should be marked by a broad pencil line on each side, with a very slight pencil tint between them, where the reflection is seen. This must be darkened wherever the reflection is lost in consequence of the vein being in some other plane than that at right angles to the line of vision, *e.g.*, when passing over some prominence, or receding into the physiological cup.

The branches of this vein and the artery accompanying it should then be drawn in like manner, the artery being represented by paler pencil lines and its central reflection being left white. Great care must be taken to depict accurately the relative width of each vessel, both on the surface of the disc and beyond its edge. The width of the vein first drawn, must also be compared with the size of the cup and the disc generally, and this vein taken as a standard with which to compare the others ; in this manner the precise representation of the size of the various vessels—a very important point—is much facilitated. Each of the other vessels should then be drawn in the same way ; as a rule, each different vein first and then its artery.

It is necessary to indicate many features by some arbitrary signs, or by reference-indications to words written on the margin. Among the points to which attention should be given, is the presence of white lines along the vessels, due to the tissue of the wall. It will be remembered that, when we speak of the vessels we are drawing, we mean only the columns of blood within them ; we cannot see the vessels themselves except in the appearance now referred to.

These white lines may be indicated conveniently by dotted lines outside the darker ones. Where the vessel becomes indistinct, there should be, of course, a corresponding indistinctness of the lines representing it; but often this is not enough to indicate the degree of concealment, and then lines may be drawn across the vessel.

The shape of the central cup should be carefully attended to, as well as its depth, and the course of the vessels down its side. The latter, together with the change in their aspect, represents the steepness of the side, and shows it at once to one who is used to ophthalmoscopic examination. The manner in which the vessels disappear at the bottom of the cup varies, and must be carefully indicated in the sketch. Often they gradually pass from view as they penetrate the tissue, which at length conceals them; then they not only become fainter but also narrower, because at the edge of the column the depth of blood is less and its tint is less deep; hence the margin becomes first concealed, and the vessel seems to lose in width as it loses colour.

In normal discs it is common to have a little softening of the edge where the chief vessels cross above and below, striated in character, and due to the large number of nerve-fibres which cross the edge there. This may be indicated by faint lines across the edge.

In general it is convenient to indicate all features that are white by dotted lines. Thus the outline of a white spot or patch should be made with dots instead of by a continuous line. If there is a difference in the intensity of the whiteness in different parts of the area, it must be drawn black with the pencil, the intensity of the white tint being inversely indicated by the intensity of the pencil shading, and the fact that it is white indicated by a dotted line around it (not then indicating the position of the edge, which should be the edge of the shading), or else by a written indication adjacent to the spot or in the margin.

So, too, the sclerotic ring should be shown by dotted lines, and also the outline of a posterior staphyloma. White spots of albuminuric retinitis may be made dark, without the risk

of error, if—as should always be the case—the more finished drawing is made without any delay to permit the nature of the case being forgotten. This lessens the practical difficulty that we have to contend with—the difficulty that we have only the lead pencil to indicate the white, the black, the red, and the grey. We must indicate the grey in a large cup or on an atrophied disc as we indicate the red tint, marking the difference by words or letters on the rough drawing, and in a descriptive note on the more finished one.

Some remarks on the production of coloured drawings may be acceptable to those who have no knowledge of water-colour work. I offer the following suggestions because they embody the results of an attempt to make such drawings without any knowledge of the art.

Colour is necessary for a perfect representation of the aspect of the fundus, but care must be taken not to lose sight of form, even in its minutiæ. The knowledge of pigments that is requisite is best gained by experiment, and it is wise to obtain pigments of the proper tints, if possible, and to mix as little as possible. The "wash" is the chief thing that is needed, and this is easily acquired with a little practice. Details only need patient care. Fortunately, in the features that can only be represented in colour, the exact shade of colour is comparatively unimportant. The precise tint of the choroid varies so much, that correspondence with nature, so far as relates to the individual eye, is not appreciated, indeed is not observed by most persons, and hence a divergence from nature is equally unobserved. Of course the limits of the variations that are met with in the tint of the choroid must not be exceeded, and in the cases in which there is a choroidal change to be depicted the amount of pigment in the choroid must be carefully observed.

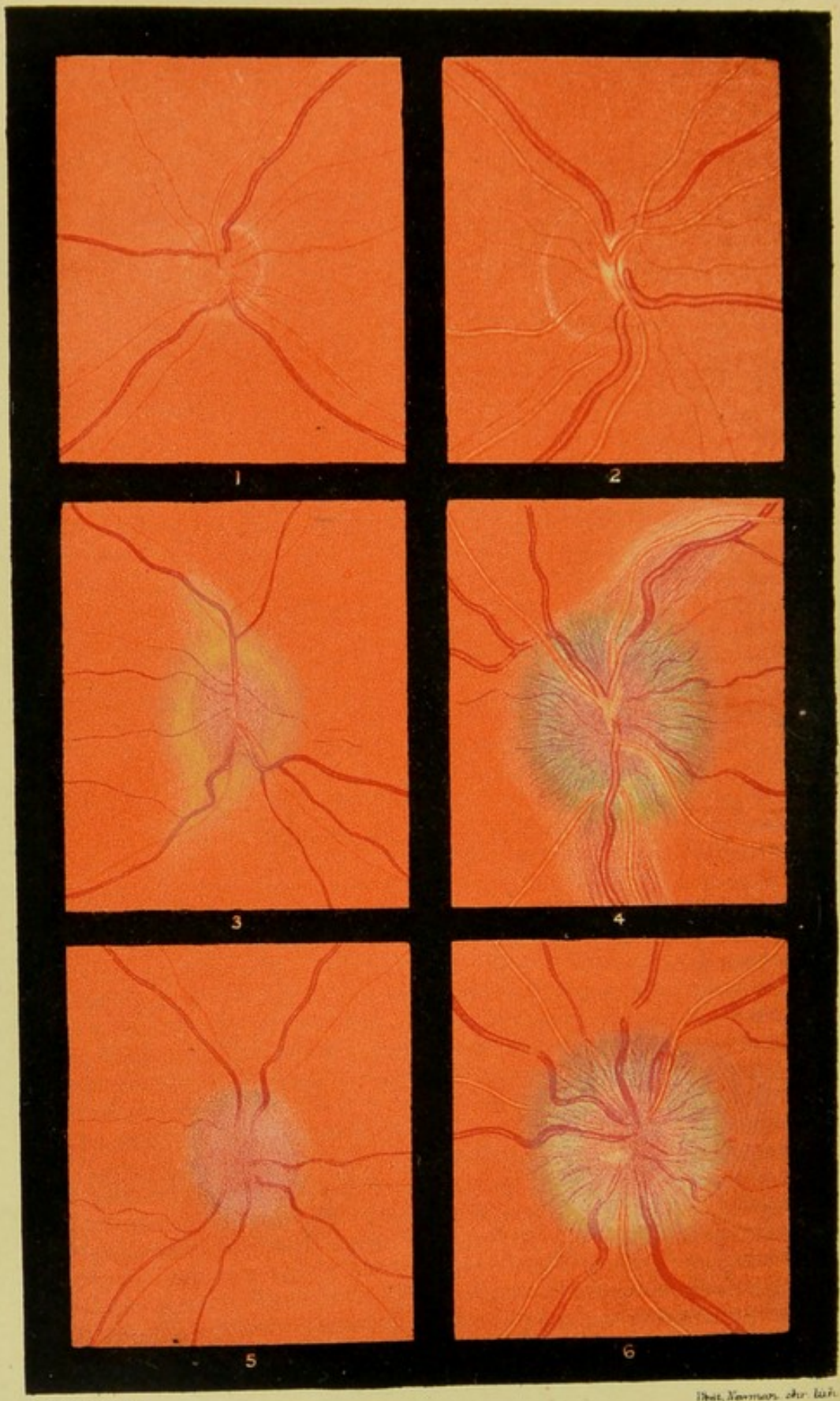
The chief difficulties with the colour are—to obtain a natural appearance as regards texture, and to obtain evenness of the tint. The beginner may take comfort in the fact that any defects will be concealed to a large extent by details afterwards added. The conspicuous forms of

the vessels prevent even a considerable unevenness from being noticed. Professional artists use Bristol board for the coloured drawings, and the surface of this, after having been well washed, is not so bad as might be imagined. I prefer, myself, a thin hot-pressed paper, damped and stretched. The grain thus obtained is finer than that of a paper which has not been hot-pressed, and yet it takes the colour perfectly well. All the sepia drawings in this work were done upon such paper. Hollingsworth's I found distinctly better than any other I tried.

The difficulties with the colour arising from these two causes—(1) getting the proper tint of red; (2) putting the colour evenly on the paper—may thus be overcome: (1) Do not use vermilion. Any colourman will give you a choice of reds sufficiently large to represent every possible tint the human blood can assume. A little light red, added to carmine, answers very well; but if the tint of any drawing which seems near nature, is taken and compared with sample tints, no difficulty will be experienced in selecting one that corresponds sufficiently closely. (2) Evenness of surface is best obtained by washing the coat of colour with water, when it is quite dry, and then placing blotting paper on it. This, of course, takes off a good deal of colour; but by laying on another coat (before the surface is quite dry), and repeating the process three or four times, a very even tint is obtained without difficulty. If any inequalities are seen they may be removed by "stippling" with the point of a brush. Some professional artists get the ground almost entirely in this manner, and it was the method adopted by the late Mr. Streatfeild, who, in his early days, made admirable ophthalmoscopic drawings. General stippling may be necessary when there are peculiarities of the choroid, for the distribution of its pigment can only thus be represented. For neuritis it is not necessary. The method of successive coats has an advantage in the softness of the edge of an inflamed disc that can be obtained. It is better to take out, from each wash, the area of the disc, by the end of a small roll of blotting paper,

than to leave it, on account of the hardness of the edge which the latter method involves (except in the hands of a skilful artist, for whom these directions are not intended). By softening the edge of the colour with a wet brush, however, then pressing blotting paper firmly on it, and repeating the process several times, any part may be made as soft in its gradation as can be desired. Sharp-edged, perfectly white spots may be produced with a penknife.

PLATE I.



W.B.G. del. ad. nat.

Hint, Newman. chr. lith.

DESCRIPTION OF PLATES.

The ophthalmoscopic illustrations contained in the following plates are from drawings of the erect image, with the exception of Pl. I. Figs. 1, 3, & 5; Pl. VI. Fig. 1, and Pl. XII. Fig. 2, which are of the inverted image. In some of the other figures, however, the drawings, although of the erect image and made on a large scale, have been reduced in the photographic reproduction nearly to the dimensions of the inverted image.

PLATE I.

Figs. 1 & 2.—*Simple congestion of the optic disc in a case of embolic softening in right cerebral hemisphere, causing left hemiplegia. Right optic disc five weeks after onset.*

Man, aged thirty, had rheumatic fever at thirteen. The congestion of the discs came on a fortnight or so subsequently to the occurrence of hemiplegia, and then remained unchanged for about six weeks. Both discs ultimately became much less congested, the left clearing first. The paralysis remained absolute, and there was rapid wasting of limbs. Later, blood and albumen appeared in the urine, and rounded hæmorrhages with white centres were found over both retinæ; soon afterwards distinct optic neuritis developed in the right eye only. Three weeks later patient died. Right middle cerebral artery plugged; corpus striatum softened throughout; traces of slight old meningitis over both hemispheres; infarcts in spleen and in kidneys, the latter being "large white;" aortic and mitral disease. The late retinal hæmorrhages were probably due to the blood-state. Note occurrence of neuritis on same side as cerebral lesion.

Fig. 1 *represents the disc as seen by the indirect method of examination.* The tint is nearly that of the adjacent fundus, and is uniformly distributed over the disc. The edge is dimly seen as a pale zone, most distinct on the right (temporal) side. The veins are large, especially one which passes apparently upwards (really downwards). Several small vessels passing from the disc are unduly visible.

Fig. 2.—*The same disc as seen by the direct method (upright image)*

—*reduced.* The uniform red tint is as conspicuous as by the indirect method. The sclerotic ring is visible on the left (temporal) side, but nowhere else is the boundary of the disc recognizable. The large size of the veins is very conspicuous, and there is white tissue about the vessels, arteries especially, in the centre of the disc, very conspicuous against the red surface.

The left disc presented nearly, but not quite, the same appearance, and after a few weeks its congestion lessened much more than that of the right.

FIGS. 3 & 4.—*Commencing optic neuritis; "congestion with œdema;" probable cerebral syphiloma. Left eye.*

The patient, aged twenty-five, had had a hard chancre at twenty; subsequently cranial nodes; an attack of right hemiplegia at twenty-three, and headache and convulsions for six months. Right optic disc full coloured, but otherwise normal. The appearance of the left is shown in the figures.

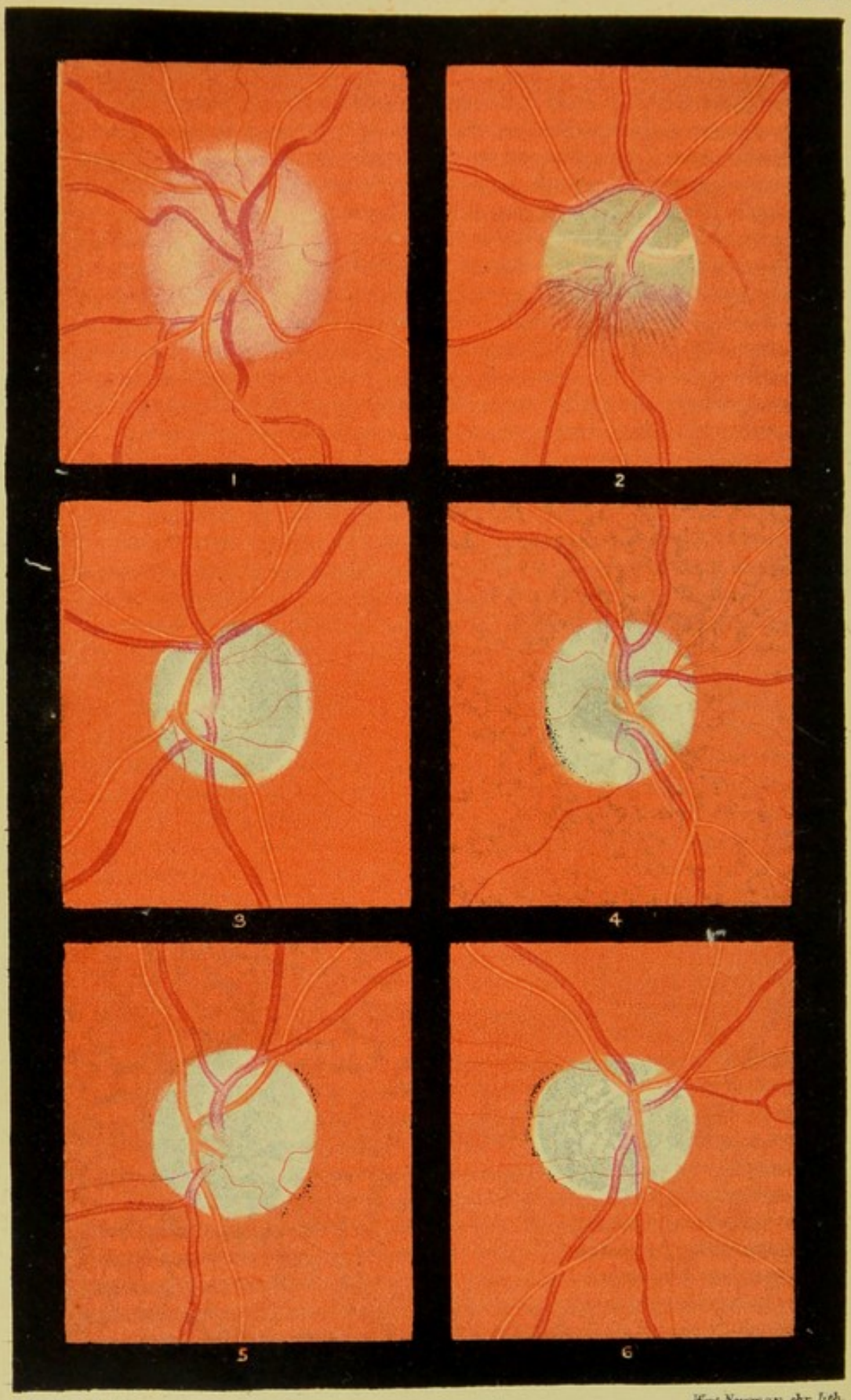
FIG. 3.—*Appearance on an indirect examination.* The edge of the disc is fairly distinct, but its surface is uniformly red—a deeper and more carmine red than the adjacent choroid. Around it is a pale halo, and this can be traced upwards and downwards along the course of the larger vessels, in the situation in which the nerve fibres are most numerous. The retinal vessels are of nearly normal size, clear to their emergence in the middle of the disc. (The arteries in the figure are rather too small.)

FIG. 4.—*Appearance of the same disc on examination by the direct method (reduced).* The edge of the disc can nowhere be seen; the pale halo is seen as a striated, reddish-grey, slightly prominent opacity, completely veiling all behind it. The increased redness in the centre is the only indication of the position of the optic disc. The opacity ceases abruptly, except above and below, where a fine striation accompanies the vessels—the pale reflection recognized in the inverted image. The veins are a little larger than normal; they curve down the sides of the swelling, but the prominence being slight, the change of plane causes only a slight diminution of the central reflection. The edge of the swelling is steepest on the temporal side (to the right), and there a small vein forms a conspicuous curve down the side.

Vision $\frac{14}{14}$. Field and colour-vision normal.

FIGS. 5 & 6.—*Optic neuritis. Right optic disc of a patient suffering probably from a cerebral tumour, causing fits beginning in the right side of the face. Man aged thirty.*

FIG. 5 *represents the papilla as seen by the indirect method.* The outlines of the disc cannot be seen; its position is occupied by a roundish prominent swelling, the centre of which is red, the outer part pale, and the sloping side greyish. The veins present conspicuous curves as they course down the sides of the swelling. They cannot be traced to the middle of the swelling, their terminations



W.H.G. del. ad nat.

West, Newman chr. lith.

being concealed in the red centre. The arteries cannot be recognized on the swelling, being visible only beyond its edge, where they have a normal course. (In the figure they are rather too narrow.)

Fig. 6.—*The same disc as seen by the direct method of examination (reduced).* Its prominence is less conspicuous, but is indicated by the curves of the veins, and so great was it that, although the fundus was distinct without a correcting lens, the top of the swelling could only be seen with + 2 D. The centre is red, and presents a fine stippling; the outer part reddish-grey, striated. The veins are larger than normal, and being numerous, are no doubt considerably enlarged. Over the prominence of the swelling their reflection is bright; but it is lost, and the vessels appear dark, as they pass down the sides of the swelling. Beyond its edge several of them are partially concealed as they dip into the substance of the retina before assuming a normal course upon the fundus. Towards the slightly-depressed centre they are lost in the tissue, some, as the lower veins, gradually, others suddenly after a slight curve, in which their central reflection is again lost. One or two arteries can be traced over the outer part of the swelling, and present there a bright reflection. Others are concealed completely by the tissue, and only appear beyond its edge. Near the centre of the disc is a small oval white spot. On the right (temporal or macular) side of the disc the red of the choroid is varied by a series of paler lines, most being concentric with and adjacent to the edge of the swelling. They depend on the folds into which the retina (perhaps only its nuclear layers) is thrown, in consequence of its displacement from the edge of the choroid—partial detachment. (Compare Pl. VII. 1, Fig. 9, p. 58, and Fig. 17, p. 61.) Vision $\frac{3}{4}$, but considerable concentric limitation of the field. The condition of the discs remained the same when last seen, several months after the drawings were made.

PLATE II.

Fig. 1.—*Subsiding neuritis; commencing consecutive atrophy. From a case of local chronic meningitis, with changes (induration, &c.) in the subjacent convolution, probably syphilitic. Left eye. Woman aged twenty-five.*

After optic neuritis had lasted about three months, the vision began to fail, and was soon reduced to bare perception of large objects in the lower outer part of each field. At this time the swelling of the discs was at its height, but the injection was becoming less marked. A fortnight later, sight improved considerably in both eyes, so that she could read large type. This improvement, however, was only temporary, as a second failure occurred in the following month, leading rapidly to blindness. The discs had now become much paler and the veins smaller (the drawing was made at this

stage), and the appearances remained practically unaltered till her death a few weeks later.

The position of the disc is occupied by a pale swelling with very soft edges and depressed centre, in which a little of the redness still remains. The area of the swelling is considerably larger than the disc, and its prominence is considerable, as is shown by the curves formed by the veins as they course down its sides. Several are slightly concealed beyond the edge of the swelling. The arteries have an almost straight course. Both veins and arteries are a little more concealed at the centre. The veins, on the fundus especially, are distinctly smaller than they were in an early stage. The small vessels are also much smaller, many having disappeared, and others can only be traced as fine lines. Vision 0.

FIG. 2.—*Neuritis subsiding irregularly, clearing from the upper half of the disc before the lower, in a case of syphilitic disease of the brain. Left eye.*

The lower half of the disc is concealed by a greyish-red, striated swelling, of moderate prominence. The veins curve over it. An artery is partially concealed by it. The upper portion of the disc—rather more than half—is clear, but has a "filled-in" look, being occupied by new tissue of a pale grey tint. A large vein has a peculiar course, curving round the upper edge of the disc. The arteries are partially concealed by the new tissue; they appear narrow and indistinct, and both these and the vein are bordered by whiter tissue. Similar white tissue marks the position of several small vessels, which can scarcely be distinguished.

FIG. 3.—*Grey atrophy of optic nerve, probably from post-orbital pressure on the nerve trunk. Left eye. Woman aged fifty-nine.*

The disc presents an iron-grey tint, greenish in daylight, uniform in the outer part, but mottled at the bottom of the central cup, grey flake-like spots being separated by the white trabeculæ of the lamina cribrosa. The central cup is deep and wide. The sclerotic ring is visible in almost the whole circumference of the disc. The vessels present no reduction in size. They are distinct to their emergence. The veins join to form a trunk, which can be seen dimly as it passes down into the substance of the nerve in the central cup. Along one artery, which passes downwards and crosses two veins, white lines, indicating the position of the wall of the vessel, are distinct against the darker veins. Vision 0.

This patient was distinctly rheumatic, and had an attack of facial palsy apparently of this nature. Eighteen months later, after exposure to cold, she had complete paralysis of all the motor nerves of one orbit, and blindness of the corresponding eye. The motor nerves recovered under treatment, while the eye remained quite blind. There was never any papillitis, but the disc slowly atrophied.

FIG. 4.—*Atrophy of optic disc, of six years' duration, secondary to*

pressure on optic commissure after previous slight damage by neuritis. Right eye. Man aged thirty-four.

To indirect examination the disc appeared white, but on direct examination it is everywhere of a faint greenish-grey tint, strongly marked at the bottom of the central depression. The veins and arteries are of nearly normal size. The edge of the disc is a little irregular in shape, and the appearance of irregularity is increased by a narrow zone of atrophy of the choroid on the temporal (left) side, within which pigment is accumulated in a narrow line at the edge of the disc. This is probably a relic of the attack of neuritis, and so also is the appearance of white lines along the lower vein; the latter was much more conspicuous soon after the neuritis subsided. The disc has a "filled-in" look, the excavation being slighter than in the case of simple atrophy shown in Figs. 3 and 6, and the lamina cribrosa being invisible. Vision 0.

During the optic neuritis his vision was at one time reduced to barely counting fingers, but it afterwards improved to nearly normal during the stage of subsidence. After this his sight failed temporarily and improved again on several occasions, without there being any ophthalmoscopic change. Finally, nearly two years after the papillitis, there was rapid failure of sight, which soon culminated in almost complete blindness. (See p. 167.)

FIG. 5.—*Atrophy of the left optic nerve in a case of cerebral disease, causing left hemiplegia, amaurosis of left eye, and loss of the left half of the field of vision in the right eye.*

The disc appeared white and sharp-edged to the indirect method, but by the direct method is pale grey. The tint is pale and uniform in the outer part, and in the centre there is a deeper grey mottling within the lamina cribrosa. The edge is clear and sharp all round. On the temporal side (to the right) is a little accumulation of pigment. The veins and arteries are of normal size, and can be traced to the bottom of the large central cup. A small vessel passes on to the disc from the choroid, and thence on to the retina. Vision 0. Boy aged fourteen. The paralysis and blindness came on suddenly during a fit one year after fracture of skull. The nature of the cerebral lesion remained hypothetical. (See charts of visual fields, Figs. 42, 43, p. 73.)

FIG. 6.—*Atrophy of the optic nerves of three years' duration in a patient presenting slight spinal symptoms (rheumatic pains in legs, darting pains in back, satyriasis, and excessive knee-jerk). Right eye.*

The optic disc is sharp-edged, the sclerotic ring conspicuous on the temporal side, and beyond it a little pigment-accumulation. The nasal half of the disc (to the right) is a soft uniform grey; the temporal half to the left is darker grey, mottled. The excavation is large, and at the bottom the lamina cribrosa is visible. Vision: quantitative perception of light only. Both discs similar.

PLATE III.

FIGS. 1 & 2.—*Right and left optic discs; caries of sphenoid bone, with secondary meningitis.* (See p. 179.)

FIG. 1.—*Right optic disc.* Characters normal. Outline clear; central cup deep; vessels lost to view as they pass down its sides. The termination of the vein can be dimly seen in the middle, beneath the nasal edge of the hollow. (Vision normal.)

FIG. 2.—*Left optic disc.* Well-marked neuritis. Edge of disc invisible; concealed by a reddish-grey swelling, which extends beyond the normal limits of the disc. The central cup is encroached upon but not quite obliterated, a small area of white reflection from it being still visible. Vessels of normal size. The veins emerge from the central depression; one, which passes directly upwards, being partly concealed at its emergence; they present conspicuous curves and lose their central reflection as they course down the sides of the swelling. The arteries present a straighter course, but cannot be easily distinguished upon the papilla. The degree of swelling is moderate; it presents fine striation, partly due to minute radiating vessels and partly to the nerve fibres. (Vision quantitative only.)

FIG. 3.—*Descending neuritis in cerebral tumour. Man aged twenty-four.* (See p. 159.)

Right optic disc. Outline recognizable on the temporal side, although not sharp; concealed on the nasal side. Tint, greyish-red, finely striated. Swelling distinct but slight. Veins, of normal size, lose their bright reflection on the sides of the swelling and are concealed just beyond its edge; one, which passes downwards and to the right, is concealed near the middle of the papilla by a white opaque spot. The arteries are narrow, and near the middle of the papilla are bordered by white lines. (Vision could not be ascertained.) For the microscopical appearances at a later stage, when the inflammation was greater, see Figs. 9, 12, 16, 17, 29, 30, 31, and XIV. 1.

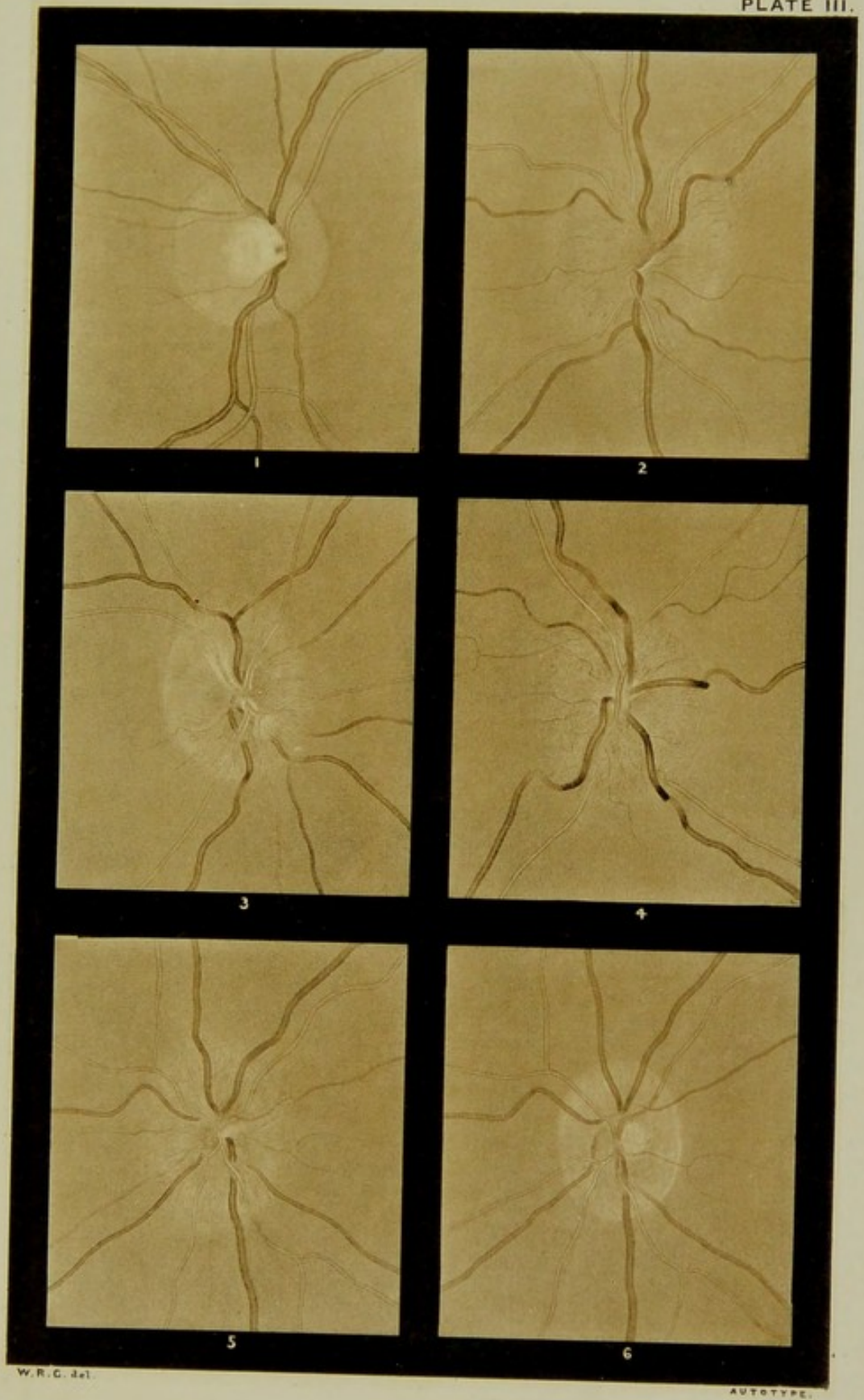
FIG. 4.—*Optic neuritis in cerebral tumour; tubercular masses in cerebral hemispheres, cerebellum, and the other eye. Boy aged eight.*

Left optic papilla. Disc concealed by very prominent swelling with a marked central depression. Veins large, and form conspicuous curves as they course down the steep sides of the swelling, some being even lost to view in their course on account of the steepness. Beyond the edge they are obscured for a short distance. Arteries partly concealed. Minute red stippling of swelling, but no hæmorrhages. Much white tissue about the vessels in the central depression. Vision: slight failure only.

FIGS. 5 & 6.—*Optic neuritis in traumatic meningitis and after recovery. Right eye.* (Case mentioned on p. 186.)

FIG. 5.—*Appearance ten days after the injury.* A pale red, striated opacity conceals the whole disc, the edge being nowhere visible; prominence slight but distinct. The central cup is not quite oblite-

PLATE III.



W. R. C. del.

AUTOTYPE.

PLATE IV.

W. R. C. del.

AUTOTYPE.

rated; its white reflection is visible at the bottom of the central depression. Veins of normal size; the upper branches, where the swelling is greatest, lose their reflection at the edge. Arteries of normal course. Vision: no evidence of impairment.

FIG. 6.—*The same disc a month later*, presenting normal characters. Edge clear and fairly sharp; sclerotic ring visible on nasal side (to the right). Central pit clear and apparently normal; steep on the temporal (left), sloping on the nasal side. The edge of the disc is seen by its relation to the vessels to be considerably within the limits of the swelling shown in the preceding figure. The vessels have a normal course. Vision normal.

PLATE IV.

FIGS. 1 & 2.—*Optic neuritis in a case of probable syphiloma of brain, and disc after recovery. Right eye. Man aged thirty-three.*

FIG. 1.—*Inflamed papilla.* Disc concealed by a prominent, red, striated swelling about twice the normal diameter of the disc. A slight central depression can be seen. The veins, not larger than normal, appear dark as they pass down the sides of the swelling. A white patch lies across and conceals one which passes downwards. On the lower part of the swelling is a small hæmorrhage. Vision: No. 1 Jäger at six inches with a little difficulty.

FIG. 2.—*The same disc three months later*, presenting very little trace of the preceding inflammation. Outline quite clear and sharp; sclerotic ring distinct; no disturbance of adjacent choroid. Central cup small but not apparently "filled-in," as the veins can be traced down its sides to their junction at the bottom. Some of the arteries on the disc are accompanied by white lines, especially one which curves downwards. A comparison of the vessels with those in the last figure will show how much they were altered in their course by the swelling. [A vein which passes upwards and to the left has by an error been drawn as an artery.] Vision normal.

FIGS. 3 & 4.—*Optic neuritis from cerebral syphiloma, and same disc after the subsidence of the neuritis. Woman aged thirty-seven.*

FIG. 3.—*Inflamed papilla.* Disc concealed under a swelling of moderate prominence, and about twice the diameter of the normal disc, concealing the veins and arteries. Colour red, and finely punctate in the centre; greyish-red and striated on the peripheral portions of the swelling. The central reflection of the veins is lost as they slope down the sides of the swelling. Veins a little larger than normal; arteries nearly of normal size. One vein, which courses from below, passes over the disc more superficially than the others and presents a double curve. There is a small hæmorrhage in the centre of the disc, and a faint white spot to the right of the centre.

Vision = $\frac{1}{11}$ and $\frac{8}{10}$. Field normal. Blind spot double normal size. (Fig. 38, p. 70.)

Fig. 4.—*The same disc two months later.* Neuritis gone; outline of disc clear in whole circumference. There is a fringe of pallor beyond the nasal edge, to the left (atrophy of choroidal pigment). Tint of disc normal, but too uniform, and the disc has a "filled-in" aspect, the two lower veins being narrowed and partly concealed by new tissue at the centre left by the inflammation. The normal central "cup" is being re-established, as shown by the curve at the central end of the lower vein; the bright reflection is lost as the vein curves down the edge of the cup; at the centre it is still almost concealed. Arteries normal.

Vision the same.

Figs. 5 & 6.—*Subsiding neuritis and subsequent atrophy (cerebral syphiloma); process of obliteration of vessels. Left eye. Man aged thirty-four.*

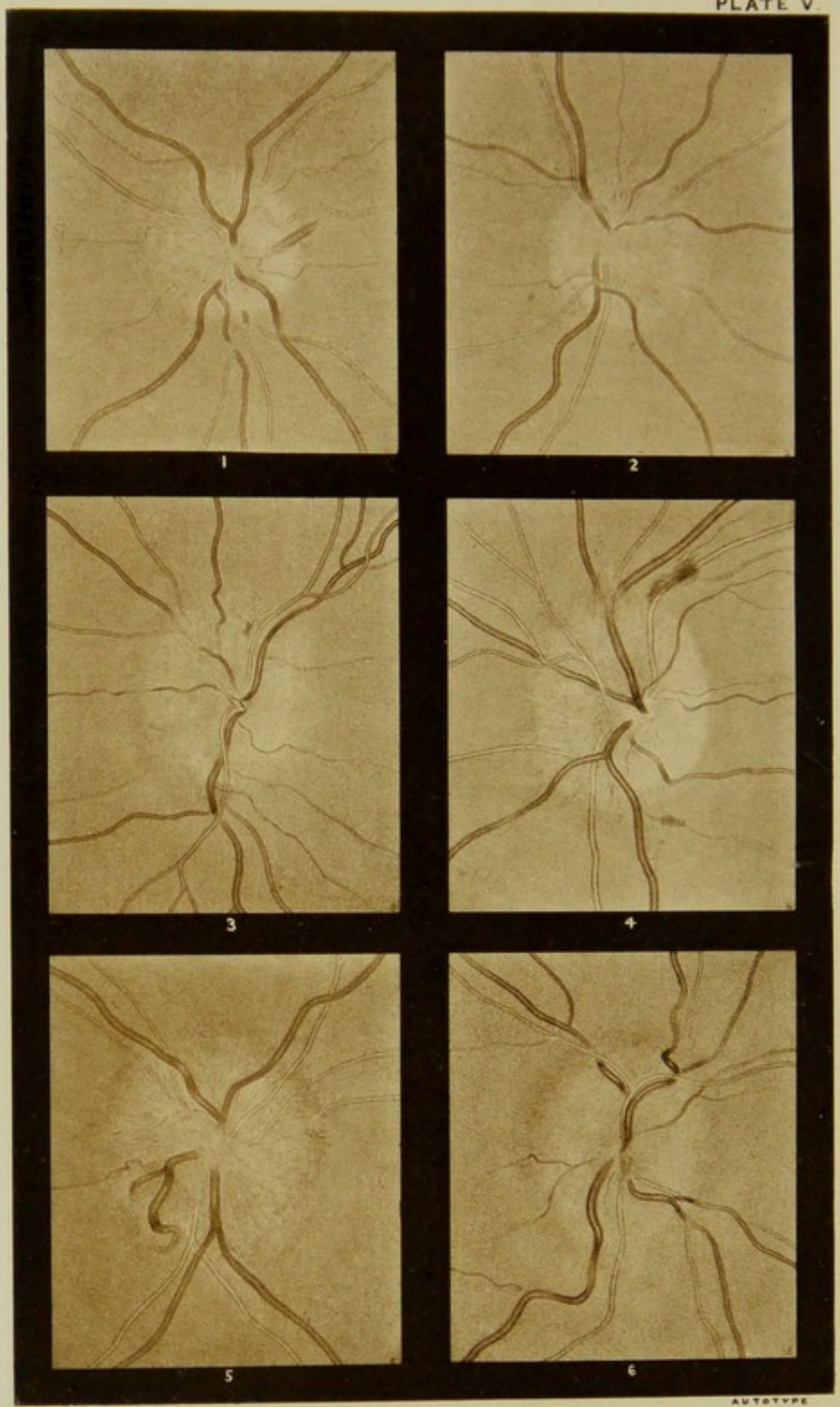
Fig. 5.—*Neuritis subsiding.* A month previously intense inflammation with hæmorrhages. Now a pale reddish-white prominence remains, with soft edges, paler in the centre than at the margin. The veins, large and dark, curve over the side of the swelling, and are obscured just beyond the edge. The arteries are small and partially concealed by the new tissue. On the surface several vessels are seen in process of obliteration. One, apparently an artery, ends suddenly at a small extravasation, and the terminal portion of the vessel is very dark, as if plugged. From the central portion of the vessel two small branches proceed.

Vision 0. Galvanic stimulation, no effect.

Fig. 6.—*The same disc six weeks later.* The swelling has subsided almost to the level of the retina; the surface of the disc is white, the centre (in the position of the physiological cup) being a little whiter than the rest. Veins and arteries are somewhat smaller than normal, the latter especially. The veins have now a straight course, and the arteries can be traced, although narrowed and obscured, to their emergence near the centre of the disc. The small vein in the other figure which had a peculiar serpentine course has disappeared. The artery, which appeared to be in part plugged, presents a very different appearance. The distal part has disappeared, and the proximal portion has dwindled in size to that of the branch, which appears to be carrying on the blood from it. Its origin from a larger trunk is now clear.

Vision: very slight perception of light; retina again sensitive to electrical stimulation.

PLATE V



W. R. G. del.

AUTOTYPE

PLATE V.

FIGS. 1 & 2.—*Optic neuritis (right and left eyes) in cerebral tumour.* The patient (in the National Hospital for the Paralysed and Epileptic under the care of Dr. Hughlings-Jackson) was a man aged thirty-five, suffering from left-sided convulsions, beginning with a visual and auditory aura (referred to the left ear), and from left hemiopia of both eyes. Subsequently coarse tremor came on in the left arm, with weakness, which gradually increased to complete left hemiplegia. The symptoms were found to be due to a tumour of the right hemisphere, in the parietal and temporo-sphenoidal lobes, extending inwards.

FIG. 1.—*Left disc.* Inner half veiled beneath a reddish striated swelling of slight prominence, sufficient to alter a little the course of the veins and partially conceal the arteries. The outer half is much less red, and its outline can be seen, but is soft. A flame-shaped hæmorrhage lies across the edge, having one extremity adjacent to a small vein. There is another small extravasation near an artery on the lower margin. Vision normal, except for the hemiopia.

FIG. 2.—*Right disc* presenting a similar appearance; the inner half concealed, the outer visible, but not clear. No extravasation.

During about six months that the patient remained under observation not the slightest change could be seen in the discs except the disappearance of the hæmorrhages. A year and a half later (two years after the drawings were made) vision had entirely failed. The inner halves of the discs were still concealed under a reddish striation, but the outer halves had become grey, without any redness.

FIG. 3.—*Optic neuritis in a case of cerebral syphilitic disease, causing left-sided weakness and convulsions beginning in the hand. Left eye.*

Disc completely concealed on nasal side (to the left), while on temporal side (to the right) the position of the edge can just be detected. Swelling moderate, altering the course of the veins, which can, however, be traced up to their emergence in the centre. The curve they present at the edge of the swelling is gentle, but their central reflection is lost there. The more abrupt backward curve presented, just beyond the edge of the disc, by a vein which passes directly downwards, is apparently determined by the position of an artery which crosses it, and which a little above this point, in crossing it again, has again depressed it. A small vein which passes upwards and to the left (in the figure) is concealed for some distance by the striated opacity. On the upper edge of the swelling is a small hæmorrhage. Neuritis bilateral. Vision $\frac{1}{5}$. Colour-vision normal.

FIG. 4.—*Optic neuritis in cerebral tumour, probably tubercular, causing left hemiplegia and hemiopia. Left eye. Girl aged fifteen.*

The temporal part of the disc (to the right in the figure) is clear,

its outline being quite distinct. Elsewhere the margin of the disc is concealed by a (reddish) striated opacity, of slight prominence. The veins are large, and those which pass downwards curve a little over the edge of the swelling, while one, which passes upwards and does not curve, is concealed at the edge. A small striated hæmorrhage lies over an artery above, the striation being in the direction of the nerve fibres. The course of the artery is not changed. Below is a still smaller extravasation upon a minute branch of a vein. Vision: No. 10 Jäger at one foot; hemiopia; all colour-vision lost. Both eyes similar. The patient improved under treatment, the disappearance of the neuritis being the first sign of the improvement. In a few weeks the aspect of the discs became perfectly normal, and has continued so now for five years.

Fig. 5.—*Optic neuritis in cerebral tumour.* Glio-sarcoma, springing from the membranes, and compressing, without invading, the right side of the pons and right hemisphere of the cerebellum, causing right-sided convulsions beginning in the hand, and afterwards left-sided attacks beginning in the face; weakness and coarse tremor in the right limbs, deafness in the right ear, and trophic changes in the right eye.

The drawing was made as the neuritis was beginning to subside. Disc concealed beneath a considerable swelling, red and striated. Veins large (beginning to lessen in size), curve over the edge of the swelling. One which passes down cannot be traced beyond the edge, where it apparently disappears. (Even when the neuritis had subsided still more, its further course could not be detected.) Arteries small, not more than one-half the size of the veins. Vision 0.

Fig. 6.—*Optic neuritis in cerebral tumour. Right eye. Woman aged thirty-three.*

The neuritis was in course of subsidence. Swelling considerable, completely concealing the disc, pale, but still reddish, darker around the margin. The veins form conspicuous curves at the edge of the swelling, one above forming a double curve in consequence of passing beneath an artery just within the edge of the swelling. All the veins are concealed for a short distance beyond the edge, and then resume a normal course upon the retina. Vision: reads No. 12 Jäger at a foot. The neuritis subsided into consecutive atrophy, sight gradually failing until vision was completely and permanently lost.

The exact nature of the intracranial condition remained obscure; there was a family history both of tubercle and cancer. Mental power soon failed greatly. The condition was similar in the two optic nerves.

PLATE VI.

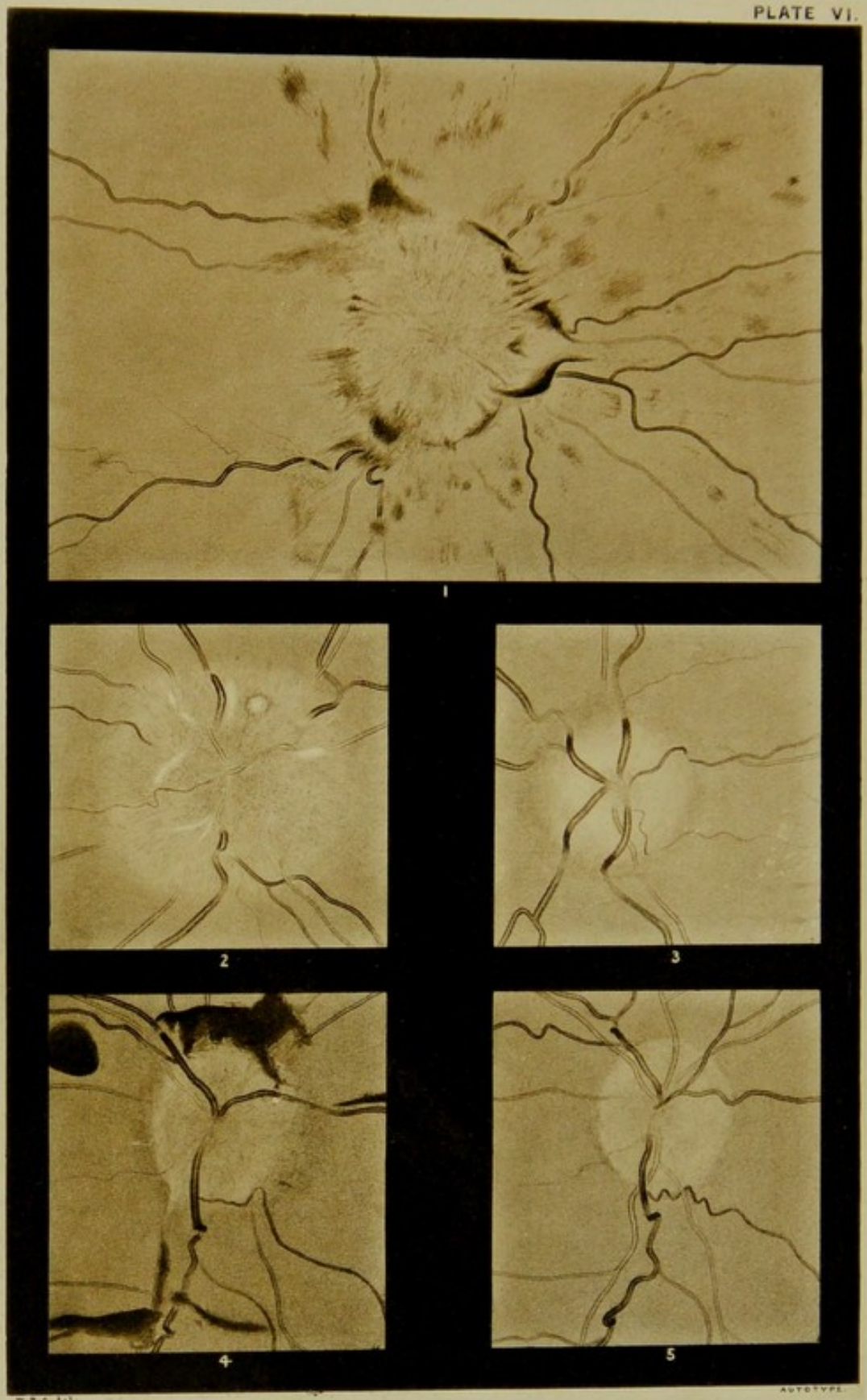


PLATE VI.

FIG. 1.—*Intense optic neuritis, with retinal hæmorrhages, in a case of cerebral tumour. Right eye. Man aged thirty-six.*

The region of the optic disc is occupied by a large swelling, in width about four times the diameter of the disc. It is irregular in outline, with very steep sides, and is bounded in every direction by extravasations. Some of these are more or less striated, others have a sharp convex edge, due to their position in the overhanging edge of the swelling. The surface of the prominence is of about the same tint as the fundus. The vessels are concealed in the substance of the swelling, except one or two, the position of which is dimly seen. Most of them appear first beyond its edge, and are then of about normal size, but at first they form conspicuous curves, the deeper portions of which are concealed. They then assume a nearly normal course. The arteries are narrow, some being scarcely visible. Numerous hæmorrhages, small and striated, are scattered over the retina in the posterior half of the eyeball, except on the temporal side (to the left). In this direction the swelling reaches almost to the position of the macula lutea, in the neighbourhood of which are many minute white dots adjacent to the edge of the swelling. Vision 0.

The patient died, but no post-mortem examination was permitted.

FIG. 2.—*Optic neuritis in a case of old fractures of the skull; inflammatory growths beneath them; at the base the results of previous meningitis. Man aged forty-nine.*

The position of the disc could be recognized by the indirect method of examination, but the edge was softened. The area of the disc was bright red, and beyond the edge was a pale halo. In the upright image the edge is completely concealed under a greyish-red swelling, of nearly three times the diameter of the disc, striated. Upon it are many white spots and lines (due to granule corpuscles, &c.), some of which correspond to the course of the arteries. One, above, is surrounded by a narrow zone of hæmorrhage. The vessels are concealed in the middle of the swelling; the veins more completely than some of the arteries. The course of the veins is very tortuous. Vision $\frac{1}{12}$. (The microscopical appearances are shown in Figs. 13, 23, 24, 33.)

FIG. 3.—*Neuritis subsiding into atrophy; slight retinal changes; tubercle of cerebellum. Left eye. Boy aged eleven.*

The disc is invisible beneath a pale, almost white swelling, depressed in the centre. Over this the veins curve. After sloping down its sides, they are concealed by the adjacent opacity of the retina for a short distance. One artery, which passes downwards, is visible on the surface of the swelling, but is also concealed beyond its edge. The other arteries appear only some distance from the edge. Midway between the retina and the macula lutea is a group of small white granular-looking spots, apparently just behind the level of a

retinal vessel which passes among them. (They slowly lessened under observation. The swelling gradually subsided, the edges of the disc reappearing and its aspect becoming that of "consecutive atrophy." Its appearance is shown in section in Figs. 49, 50.) Vision 0.

He died from meningitis, probably tubercular. At the necropsy tubercles were found in the cerebellum and medulla oblongata.

FIGS. 4 & 5.—*Subsiding neuritis, recent hæmorrhages, and same disc after recovery.*

The patient had been in the London Hospital, under the care of Dr. Hughlings-Jackson, suffering from the symptoms of cerebral tumour, and presenting intense optic neuritis. Under treatment the symptoms subsided and the neuritis gradually lessened, but during subsidence several fresh hæmorrhages appeared. He died some years later, and the brain presented softening of one anterior lobe, with the remains of an absorbed syphilitic gumma. Cicatrices were also found in the liver.

FIG. 4.—*Subsiding neuritis.* The outline of the disc can be seen, but is not clear; its surface is reddish in tint, and the swelling of the papilla is still considerable, as evidenced by the curves formed by the veins in passing over its edge. Several large extravasations are seen. One of these, below, follows the course of an artery. Another above and to the left is round, not striated, and therefore probably situated in the deeper layers and not in the nerve-fibre layer. Vision $\frac{1}{1}$; fields normal.

FIG. 5.—*The same, two months later.* The hæmorrhages have entirely disappeared. The disc is clear, and its swelling has almost subsided. But the tortuosity of the vessels has increased, probably on account of their permanent extension by the long-continued swelling.

PLATE VII.

FIGS. 1 & 2.—*Unilateral optic neuritis; probably cerebral syphiloma. Man aged forty-four.*

FIG. 1.—*Left optic disc* concealed by a swelling—reddish, striated, depressed in the centre. The veins, a little larger than normal, curve over it, and some are concealed beyond the edge. In the central depression the veins pass behind the arteries and are unduly concealed by the swollen tissue. The artery which passes upwards is visible throughout; those which pass downwards are distinct at their emergence in the depressed centre, but are concealed by the swelling, to reappear at its edge. No hæmorrhages. Just beyond the edge of the papilla is a series of pale concentric lines parallel to the edge, due to the folds in the displaced retina; they are limited above and below by a small vein. Vision: counts fingers only.

PLATE VII.

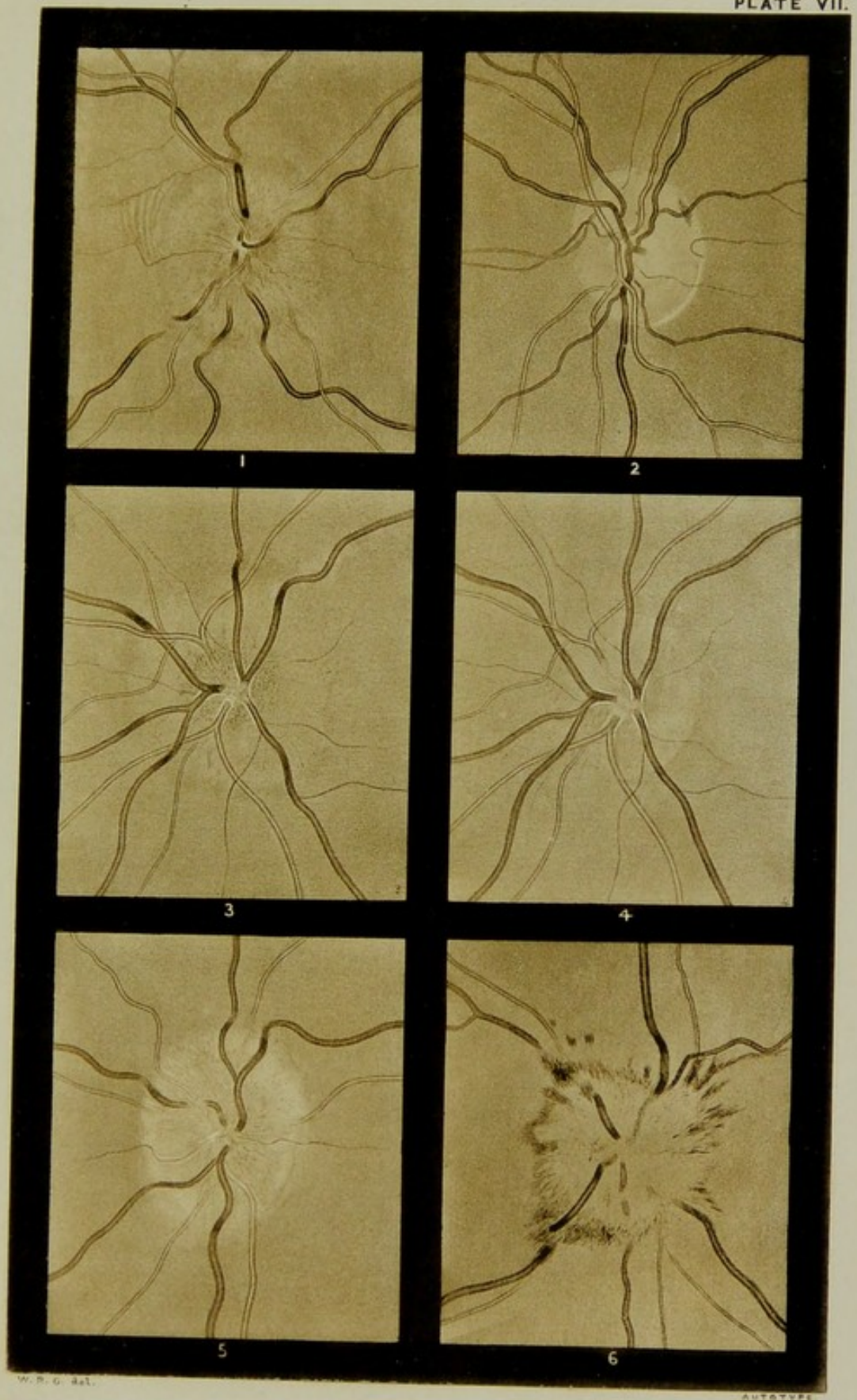


FIG. 2.—*Right optic disc* presenting normal characters. A small deposit of pigment lies across a vein.

The right disc never became inflamed, but both discs eventually became atrophied, doubtless from an intracranial cause. It is possible that both nerves were damaged in front of the commissure, and that in one only did the inflammation descend to the eye.

FIGS. 3 & 4.—*Very chronic optic neuritis, in a case of epileptoid convulsions. Girl aged fifteen.*

FIG. 3.—*Left disc.* Outline obscured by neuritic swelling of slight prominence: the centre stippled red, the periphery only slightly lighter in tint than the fundus. Veins, of nearly normal size, concealed in centre by whitish tissue, which accompanies the larger trunks of both arteries and veins for a short distance. The double contour of the veins is lost on the sides of the swelling. Vision: No. 2 Jäger, spells No. 1. Appearances unchanged during four months' observation.

FIG. 4.—*The same disc two years later.* All swelling is now gone. The outline is clear on the outer (temporal), indistinct on the inner (nasal) side. Veins large; at their junction in the disc they are even more concealed than before, and the white tissue about them is still very conspicuous. Vision, same.

FIG. 5.—*Optic neuritis in anæmia. Girl aged seventeen.*

The outline of the disc is lost under a pale, reddish-grey swelling, of slight prominence, a little larger than the disc. The veins, of normal size, lose their reflection as they curve down the sides of the swelling, and some are obscured beyond its edge as they dip into the substance of the retina. Some of the arteries are concealed; others distinguishable with difficulty. There is a small white spot near the centre of the swelling. Vision (uncorrected) $\frac{1}{8}$.

Eyes hypermetropic. Both discs cleared and vision became normal. A few months later there was a temporary return of the anæmia and of the papillitis, but vision remained normal. (See p. 243.)

FIG. 6.—*Optic neuritis in a case of lead poisoning, with cerebral symptoms. Man aged forty-five.*

The disc is concealed by a swelling of moderate prominence, bordered by a fringe of striated hæmorrhage, and of a colour nearly that of the fundus. Veins a little larger than normal. Arteries concealed by the swelling, and most of them very narrow on the retina.

His vision was considerably impaired, but could not be accurately tested, owing to his mental state. (See p. 272.)

PLATE VIII.

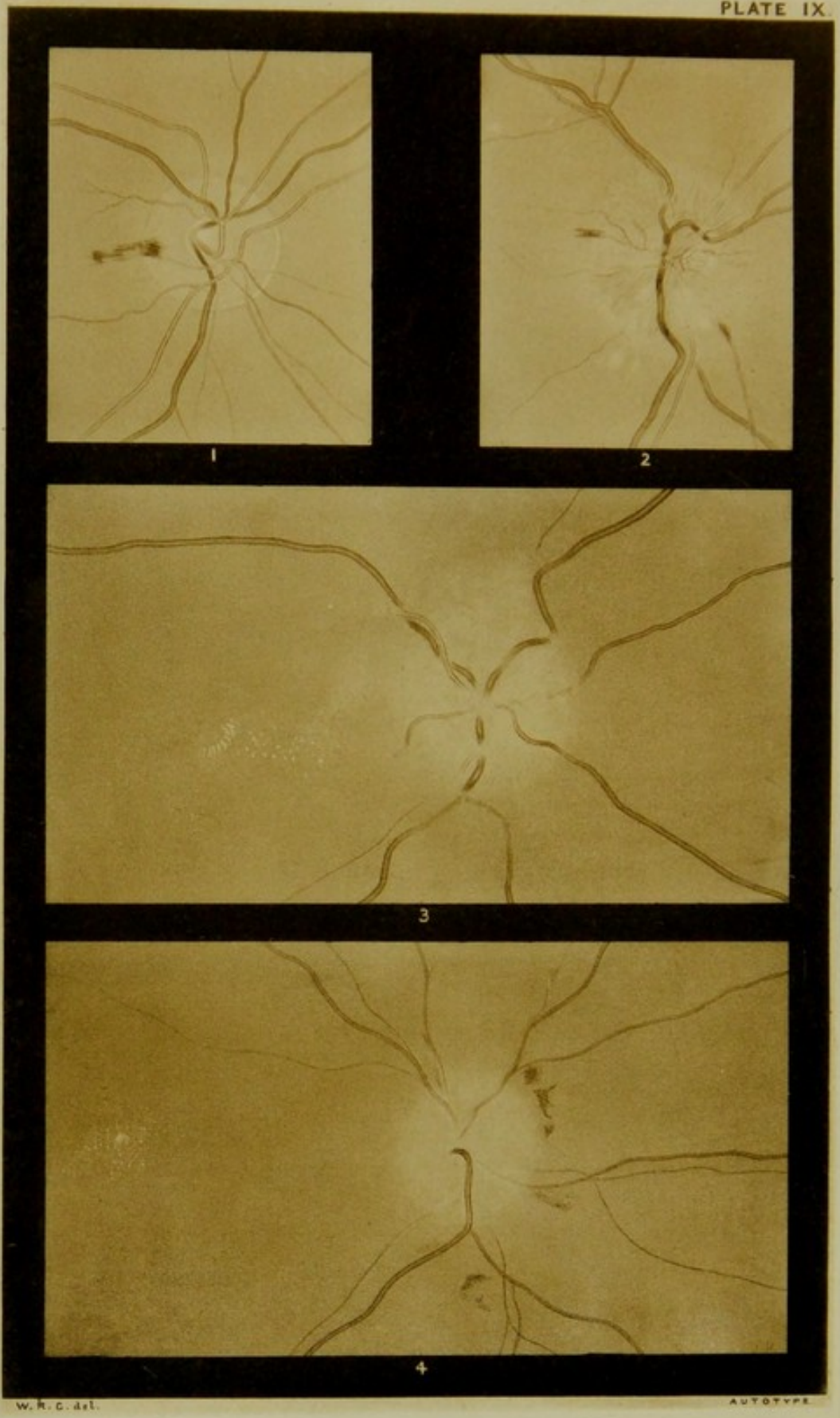
FIGS. 1 & 2.—*Intense neuro-retinitis, probably idiopathic, in a chlorotic girl, leaving changes simulating albuminuric retinitis.*

FIG. 1.—*Right fundus oculi during the height of the neuritis.* The papilla presents a very large pale red swelling, five times the transverse and six times the vertical diameter of the disc. The peripheral portions are paler than the central. Its sides are steep, and marked by scattered striated hæmorrhages. Even the tortuous veins are almost completely concealed by the swelling, the highest parts of their curves alone being seen. At the edge all reappear, are greatly distended, and form conspicuous curves, most of them being again lost for a short space in the retina. The arteries are all concealed. Many extravasations fringe the swelling. The largest lies over a vein which passes downwards: it is striated, and has a paler centre. The pale edge of the swelling is irregular, presenting several projections, and beyond it are many pale spots in the retina. The swelling on the temporal (left) side reaches as far as the macula, and just beyond it is a group of white, rod-shaped spots, arranged in a fan-like manner, and evidently situated on the temporal side of the macula. There are a few small hæmorrhages here and there in the fundus beyond the limits of the swelling. Vision: No. 19 Jäger; considerable limitation of field, especially upwards and inwards. Loss of colour-vision except for red.

FIG. 2.—*The same fundus three months afterwards.* All the swelling has disappeared. The disc is clear, but has a "filled-in" look, the vessels being partly concealed at their emergence. Both arteries and veins are very narrow. The extravasations have disappeared; the white spots in the retina persist, but have a more granular aspect. Some extend along the vessels, and one or two have an irregular linear course as if corresponding to the position of choroidal vessels. Many white areas lie in the part of the retina around the disc which was formerly occupied by the swelling. The fan-like group of spots, adjacent to the macula, has become still more conspicuous, and others appear adjacent to them, and of similar arrangement; so that the aspect of albuminuric change is very closely simulated. Vision: quantitative perception of light only. (See p. 244.)

W. R. C. del.

AUTOTYPE


1
2
3
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W. R. C. del.
AUTOTYPE

PLATE IX.

FIG. 1.—*Hæmorrhage on optic disc in a case of renal disease, arterial disease, and acute cerebral lesion. Right eye.*

The optic disc is otherwise normal; the central cup distinct, narrow but deep; the arteries and veins of normal size. On the temporal side of the disc is a small extravasation, striated, extending on the retina about a disc's breadth. It has apparently arisen from the rupture of a small vessel, which can be traced to, but not beyond, the hæmorrhage. It had given rise to no symptoms.

FIG. 2.—*Neuritis albuminurica. Right optic disc of a man suffering from chronic renal disease, convulsions, and mental derangement.*

The disc presents the signs of slight but distinct neuritis. Its outline can be nowhere seen; there is slight swelling; the tint of the papilla is red, and the redness is striated. Many small vessels radiate from it on to the retina—more than is common in neuritis. The veins are rather large. The arteries are very narrow—not more than one-half the diameter of the veins. One small hæmorrhage exists on the temporal (left) edge of the disc. On the surface of the papilla are several white spots, irregular in shape. One is situated over an artery, another near the middle of the disc, and one near the lower edge. One small soft whitish spot can be seen on the retina near a vessel above the disc, but this is the only trace of retinal affection. (There were no spots near the macula lutea.) Vision: No. 12 Jäger.

FIG. 3.—*Albuminuric neuritis in a man suffering from chronic renal disease (granular kidney), intense headache, and who died shortly afterwards of uræmia. Right eye.*

The disc is concealed by a considerable greyish-red swelling, stippled and striated. The veins are concealed at their point of emergence, curve over the prominence, and are again concealed at its edge. Beyond, they have a normal course and size upon the retina. The arteries, where visible upon the papilla, are a little below the normal size; but beyond, upon the retina, they are much smaller than normal, some being scarcely visible as mere lines, and two cannot be detected beyond the edge of the papilla. There are a few very minute shining white spots upon the centre of the swelling; between it and the macula are several white flecks, and close to the macula a few radiating dots and lines are arranged in a fan-like form. Vision: reads No. 6 Jäger. (See p. 98.)

FIG. 4.—*Subsiding albuminuric neuritis. The fundus of a patient suffering from chronic Bright's disease (probably granular kidney), with a pulse of very high tension.*

The papilla is slightly prominent, greyish-white, the edges of the disc being concealed by it. The veins are narrow and the arteries extremely small, recognizable only in narrow lines. One or two small extravasations are seen near the disc, and farther off are several small collections of pigment, probably the remains of former extravasations.

PLATE X.

FIG. 1.—*Acute nephritic retinitis, in a patient suffering from chronic renal disease, consecutive to an acute attack twelve years previously. Man aged twenty-one.*

The disc is veiled by a pale opacity, not prominent, which extends on to the adjacent retina. Many soft white areas and striated hæmorrhages are scattered over the posterior segment of the retina. The veins are a little larger than normal. Many of them are much concealed at the edge of the papilla. The arteries are large and conspicuous over the disc, but cannot be traced beyond (probably because they become contracted in size, and are concealed by the retinal opacity). Vision $\frac{1}{12}$. For the microscopical appearances see Figs. 68, 69, 70, 72.

FIG. 2.—*Chronic retinal changes in albuminuria, from a case of acute renal disease passing into the chronic form. Right eye. Woman aged twenty-four.*

The disc and its central cup are normal. The vessels have a normal course. Many irregular white spots lie around the disc, especially between it and the macula, around which is a halo of small spots, for the most part very minute; one or two larger and very white. The other spots are soft-edged: some of them are superficial to the veins. There are a few small hæmorrhages, most of them adjacent to white spots. A small vessel which passes upwards is accompanied by extravasation, as if into its perivascular sheath. Vision: reads No. 12 Jäger.

PLATE XI.

FIG. 1.—*Retinal changes in a case of progressive pernicious anæmia. Right eye. Man aged forty-seven.*

The general tint of the fundus is paler than normal. The disc is clear and the vessels distinct almost to their origin in the centre. The veins are very broad and pale, scarcely darker than the arteries. Their central reflection is broad and indistinct. The arteries are rather narrower than normal, and very narrow in proportion to the veins. A large number of striated hæmorrhages lie around the papilla. Many of these are adjacent to vessels, in front of or beside them, but the course of the vessels is not disturbed. Some white spots are seen, most of which are adjacent to extravasations, one or two being surrounded by a halo of hæmorrhage. One large white spot above the disc has an irregular extravasation below it, but only a few small spots of blood above it. (See p. 245.)

FIG. 2.—*Retinal changes in leucocythæmia. Right eye. Man aged twenty-seven.*

The optic disc is clear. The course of the vessels is normal. The retinal veins are very broad—at least twice their normal width. Their central reflection is in some veins narrow and indistinct, in

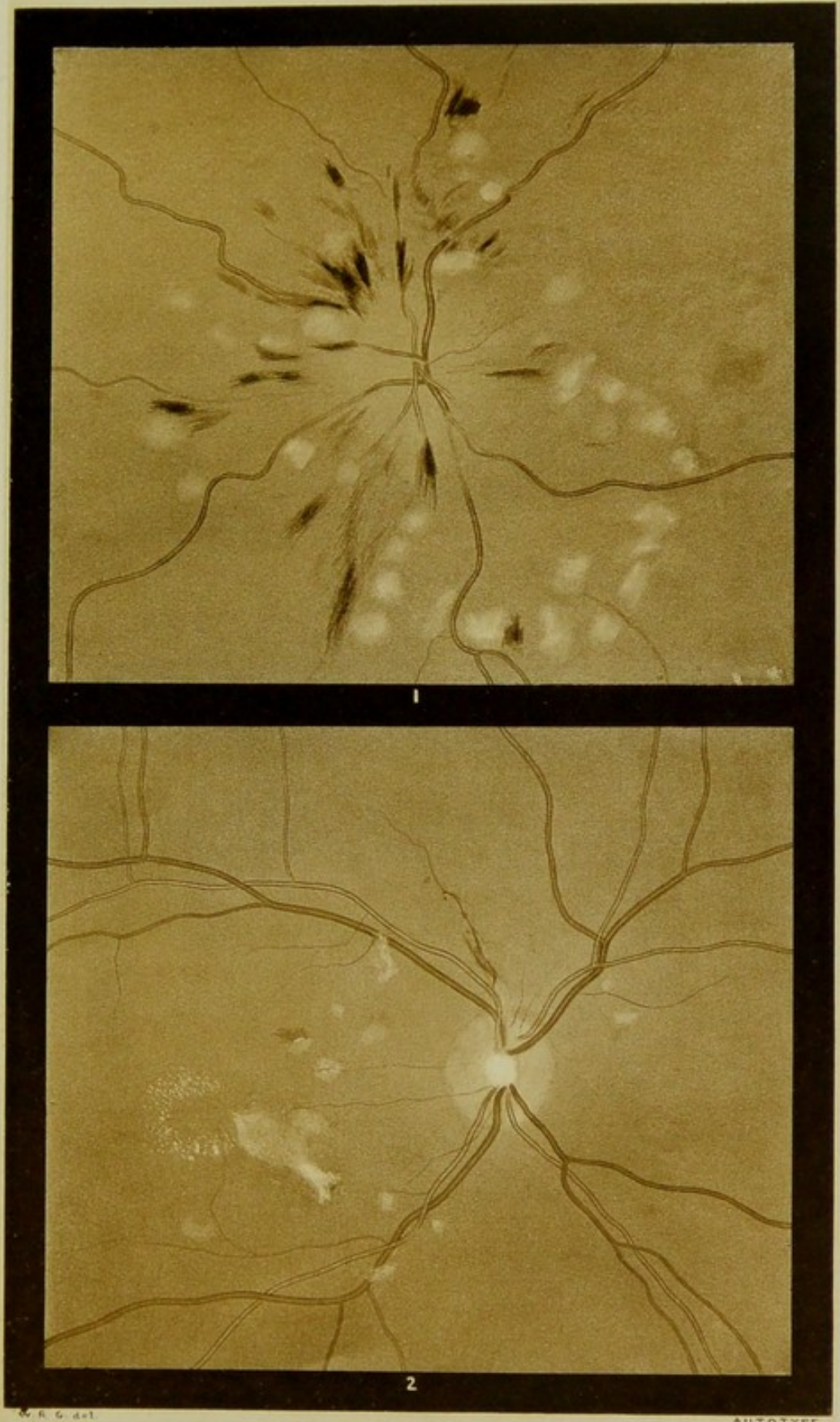

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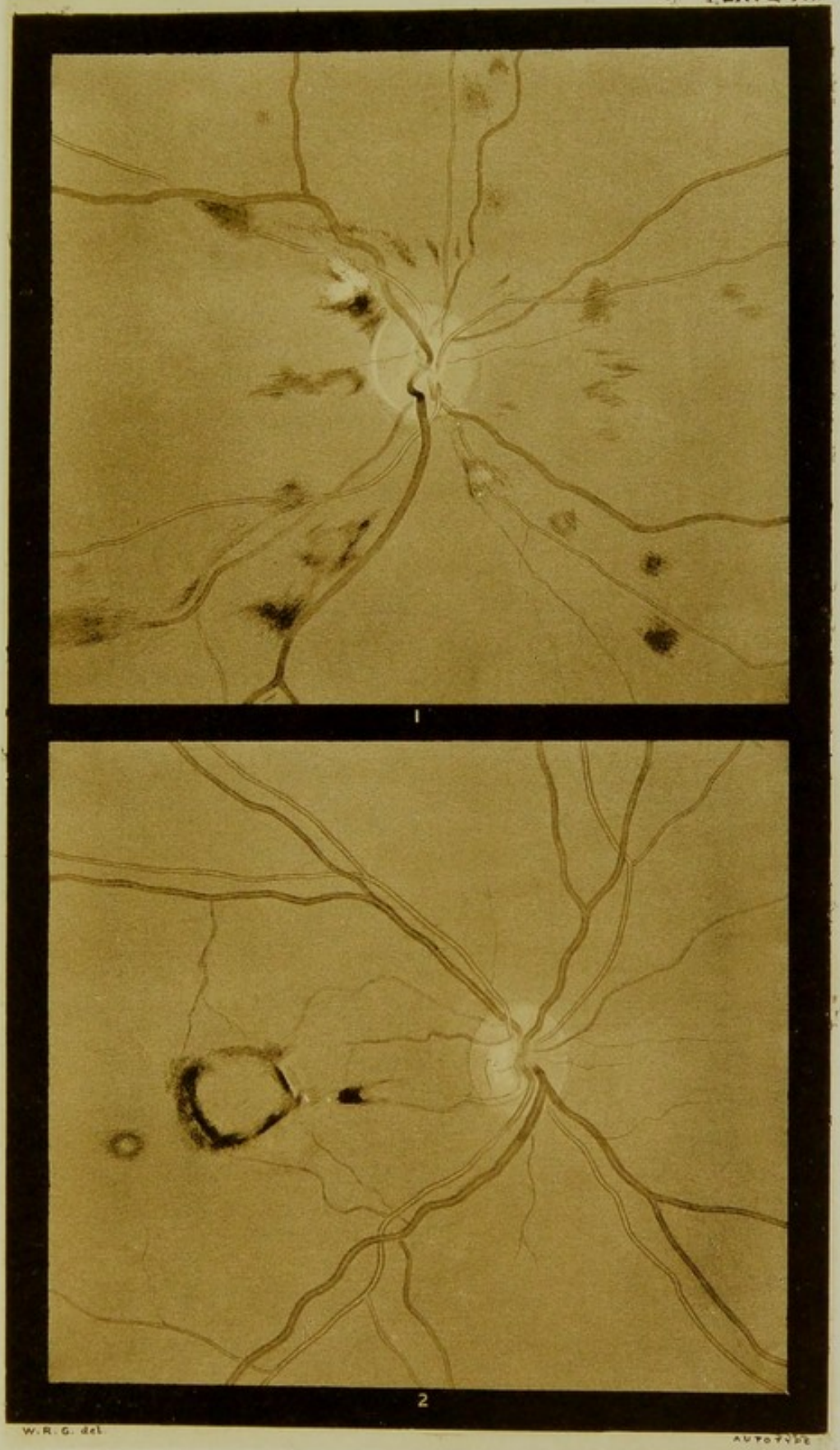
PLATE XI.



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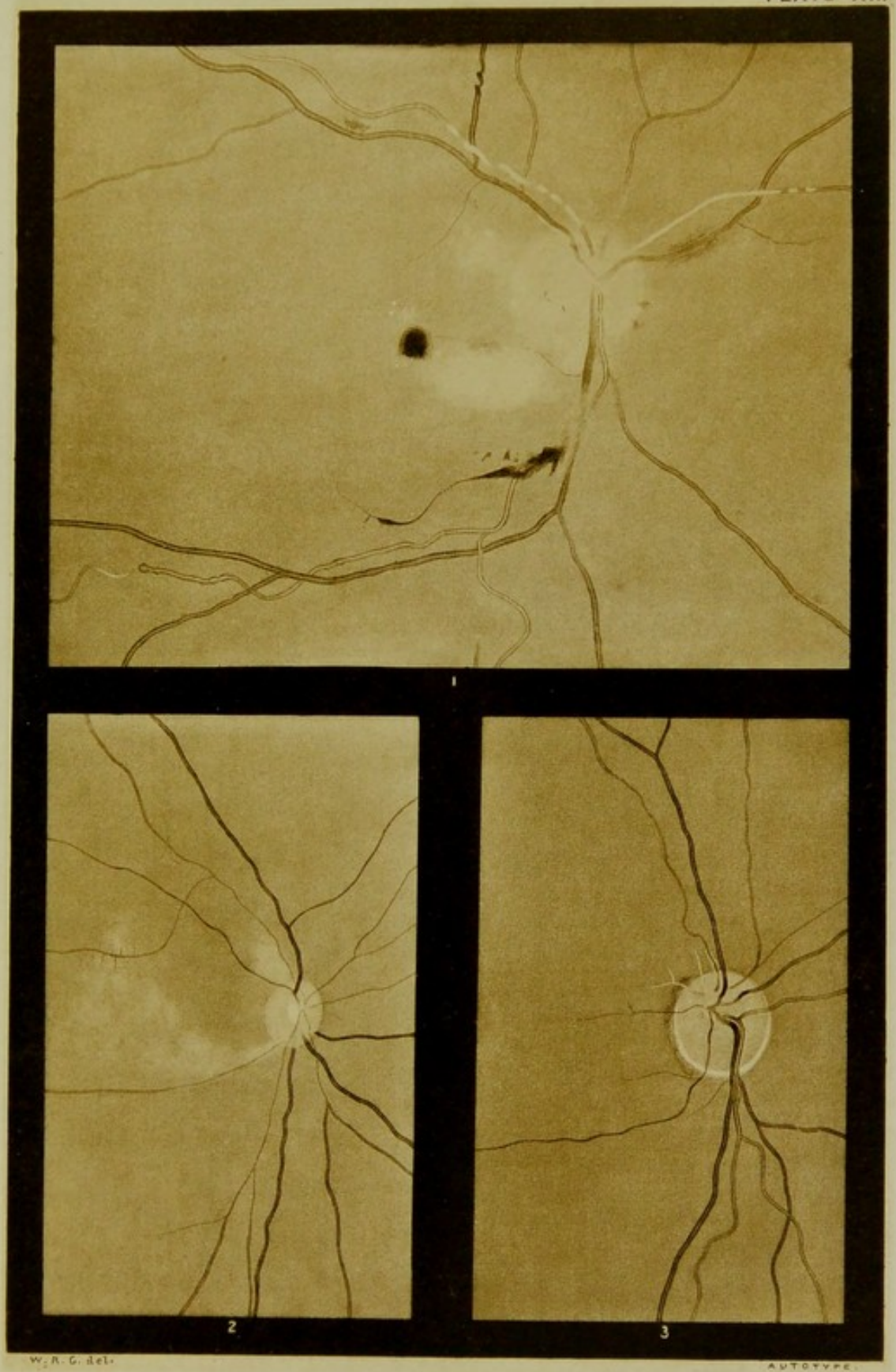

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others it is broad. The disproportion in size between the arteries and veins is thus very great. The veins are exceedingly pale, scarcely darker than the arteries. An annular zone of hæmorrhage surrounds the macula lutea, broader on the temporal than on the nasal side. On the latter, adjacent to it, the retina presents a grey reflection. Between this and the disc is a striated hæmorrhage in which are one or two white spots. On the outer side of the annular extravasation is a small, soft, white spot surrounded by a halo of hæmorrhage. The extravasation had caused a corresponding central defect in the field of vision. (Subsequently the veins became still larger and more tortuous as in Fig. 2, p. 11.)

PLATE XII.

FIG. 1.—*Retinal changes (perivascular disease, aneurisms, &c.) in a case of chronic renal disease. Right eye. Woman aged thirty-six.*

The outline of the optic disc can be seen on the nasal (right) side, but is not very distinct. Its temporal portion is concealed by a white opacity, which extends on the adjacent retina towards the macula lutea. Near the latter are a few minute white spots. Several small extravasations are seen: one, rounded in form, near the macula, and another below, which extends for a long distance along the course of a small vessel, wider at parts than at others, and in one place interrupted. Another extends, as a linear extravasation, along the course of a vein which passes directly downwards. Three arteries which pass upwards present a peculiar appearance, being concealed more or less completely by white bands, corresponding in width to the vessels. One, which passes upwards and to the right (in the drawing), is masked for a considerable distance by such a band, which ceases suddenly, and, before its termination, presents two interruptions. The vessel beyond this sheath, and in the interruptions, is seen to present perfectly normal characters. Another artery, which passes upwards and to the left, is free at its origin, but just beyond the edge of the disc is concealed by a similar band. It pursues a somewhat wavy course, the lower parts of the curves being indistinct. Like the other, the band ends abruptly, and the vessel beyond presents a normal appearance. Another artery, which arises in the disc from that last described, has a similar white sheath from its commencement to its disappearance behind a vein. It emerges some distance beyond, free. A vein passing upwards presents peculiar corkscrew-like curves. The vein which passes downwards is invisible for a short distance, beyond the extravasation just described, together with its accompanying artery. The arteries are, for the most part, otherwise normal, but one, which passes directly downwards, presents, some distance from the disc, several —at least four—distinct dilatations, evidently minute aneurisms. The central reflection from the vessel broadens out in these dila-

tations. The last one is globular, and appears at first sight to terminate the vessel, but closer inspection reveals a narrow white band passing from it, which farther on broadens, and gives origin to a branch of an artery of normal appearance. Here and there in the retina are small collections of pigment. Vision: counts fingers only.

FIG. 2.—*Embolism of the central artery of the retina, occurring simultaneously with an embolism of the middle cerebral artery. Left eye, indirect image. Man aged thirty.*

The drawing was made about a fortnight after the occurrence of the embolism. The disc (previously veiled by opacity) is clear and pale (not quite pale enough in the figure), the peripheral part almost, but not quite so clear as the central cup. Its edges are sharp. The veins have a normal size and course. Several of them, however, disappear at the edge of the disc. The arteries are filiform on the disc and for some distance beyond. Some remain, as far as they can be seen, narrow (even to the periphery of the retina); others become wider at a distance from the disc which varies in the case of different branches. From the upper part of the disc a white opacity extends a short distance on to the retina. A similar but narrower white area extends from the lower part of the disc, being evidently situated behind the level of an artery; it gradually widens and becomes less intense, and is continuous with a mottled opacity which occupies the region of the macula, and is the remnant of a large white area which at first occupied this region. A branch of an artery which courses across the upper part of this area is evidently dilated, and the minute branches which come from it are abnormally distinct. Vision 0.

For the microscopical appearance of the embolus in the retinal artery, see Fig. 4, p. 36.

FIG. 3.—*Partial embolism of the central artery of the retina. Right eye, direct image. Woman aged twenty.*

The disc is clear; the central cup and sclerotic ring distinct. The veins are of normal course and character. One division of the central artery, comprising the branches which course downwards and to the right (in the figure), is perfectly normal. The branches of the other division emerge from the upper part of the disc. Of these, two which pass upwards and outwards (to the left) are completely obliterated, visible only for a short distance as white threads. Two others which pass upwards are very narrow, but the central reflection can just be distinguished. One of them is accompanied for a short distance by fine white lines along its sides. Both vessels, some distance from the disc, become wider and resume their normal appearance; a branch of one which passes to the right remains filiform throughout. No changes visible in the neighbourhood of the macula. Vision: the field presented a defect corresponding to the area supplied by the obstructed vessels. (See Fig. 5, p. 39.)

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